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OF
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AND
LARYNGOLOGY

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I

FIFTY YEARS OF OTOLARYNGOLOGY IN AMERICA

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ST. LOUIS

In reviewing the progress of otolaryngology in America during the last fifty years it is impossible to mention individually the scientific accomplishments of the members of this profession. Such a review would fill a large volume. Rather an effort will be made to relate the progress that has been made in the profession as a whole. In part this can be accomplished best by comparing certain things as they existed fifty years ago with what they are today.

Fifty years ago the practice of otolaryngology outside of the metropolitan centers was mediocre. A doctor could be a dermatologist today; or overnight he could change the sign on his office door and the next day he would be an otolaryngologist. Such a combination as diseases of women and the ear, nose and throat existed. Very excellent otolaryngologists in metropolitan centers had an office in the city for ear, nose and throat work and an office in a suburb for general practice. Otolaryngology in America was in its infancy. It had no scientific background. It was the age of nasal spraying and medication of chronic discharging ears. Almost every patient was furnished with a chest one-fourth as large as a steamer trunk. In one corner of the chest was a pump to compress air, a reservoir for the air and a series of bottles containing various drugs in oil. The practice of otolaryngology in this manner was not limited to the man in the small town.

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Little surgery was done. Mastoid operations were performed only when there was a serious complication or a manifest acute abscess with swelling. I remember one very prominent surgeon depending upon the use of boric acid drops over a period of months or years rather than the drainage of the mastoid abscess. This condition was brought about by lack of facilities for the proper teaching of otolaryngology in this country.

The surgery of the ear, nose and throat was developed during the early part of the last fifty years. As one would expect the pendulum swung too far the other way and needless operations on the nasal sinuses particularly were performed. Many patients were made worse instead of better. Then came the work of clinical anatomists, as Shambaugh and Mosher, and clinical physiologists, as Sluder, Proetz and Fenton. Only a very few of the outstanding anatomists and physiologists are mentioned. Allergy in its relation to diseases of the ear, nose and throat was emphasized by Hansel and others. The relationship between diseases of the nasal sinuses and systemic conditions, particularly nutrition, was stressed. As a result of all these studies otolaryngology was placed on a sane and rational basis. The number of operations particularly on the nasal sinuses has been tremendously reduced.

Three factors have produced changes in the last fifty years that have made otolaryngology a major specialty and put the practice of it on a scientific basis. First has been the development of the graduate teaching of otolaryngology, second the dissemination of knowledge by the otolaryngological periodicals and third the work of the American Board of Otolaryngology.

During the last fifty years there have been great changes in the undergraduate teaching of this subject. In most medical schools fifty years ago no correlation existed between the teaching of the specialty and general medicine and pediatrics. In the clinic the ear, nose and throat were considered separate from the rest of the body and so studied. The idea was to teach the practice of otolaryngology. Today the effort is made to so prepare the undergraduate that when he has his residency he will be able to study the ear, nose and throat and correlate his findings with those of general medicine. He will be prepared to do those things which the general practitioner must do. He will know the influence of otolaryngological conditions on the body as a whole. He will also know the influence of diseases of organs far removed from the head upon the ear, nose and throat.

In many schools today far fewer hours are devoted to the teaching of otolaryngology than fifty years ago. However, the work is

condensed; the teaching is such that the student can use his knowledge in his ward service in medicine, pediatrics and surgery. In short he is prepared to do the work in diseases of the ear, nose and throat which the general practitioner should do.

The general practitioner today is not required to treat or operate on otolaryngological cases. The exigencies of the situation have been met by the great increase in qualified otolaryngologists, increase in population, hard surface roads, the automobile and county hospitals.

The graduate teaching of otolaryngology fifty years ago was very mediocre. Short courses were given in graduate schools. I doubt if there was one residency limited to otolaryngology in the country.

After the World War the leaders of the otolaryngological profession became very much interested in the development of the specialty. They naturally turned to the postgraduate teaching of otolaryngology. The American Otological, Rhinological and Laryngological Society appointed a permanent committee to investigate the facilities for the postgraduate teaching of otolaryngology and to aid in its development. This committee had much to do with the production of well trained otolaryngologists.

The American Academy of Ophthalmology and Otolaryngology established a teachers' section. This met once a year. All important otolaryngological teaching problems were discussed. This section has developed so that it exerts today a very large influence upon the teaching of otolaryngology.

The American Board of Otolaryngology has been a potent factor in the development of this specialty. It has influenced the graduate teaching of otolaryngology. It has made every teacher of this subject review his work and see how it meets the standards set up by this board. Every residency in ear, nose and throat has been benefited by the activities of this board. Due to the work of the board and the interest of the special societies the graduate teaching of otolaryngology in America has become the best in the world. There is no reason why anyone should go abroad in order to become a most competent otolaryngologist.

Otolaryngologists have not confined themselves to the surgery and treatment of diseases of the ear, nose and throat. All are helping in the management of the handicapped child. Influenced by the work of Urbantschitsch in Vienna and Hudson Makuen in this country they are helping with the education of the deaf child and the correction of speech defects.

II

THE ANNALS: 1892-1941

ARTHUR PROETZ, M.D.

ST. LOUIS

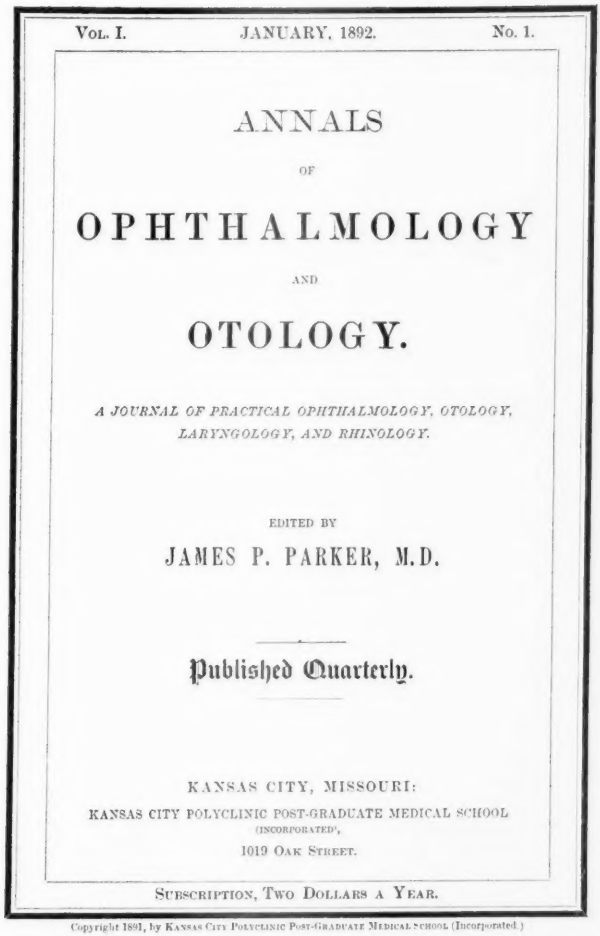
The history of the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY is the history of otolaryngology reflected in a peculiarly relevant manner. From the beginning its articles appear to have been selected with conservatism and judgment, and there is little to be found in its pages that has to do with the visionary and the transient.

This Journal first saw the light in January of 1892, and the place of its nativity is Kansas City. It first appeared as the Annals of Ophthalmology and Otology under the editorship of James Pleasant Parker, M.D., who received all communications and "$2.00 per annum in advance," at the southeast corner of Twelfth Street and Grand Avenue.

According to the prospectus it was believed that "the time has come when the busy, progressive practitioners of medicine in the Western and Southern states will give substantial support to a Periodical devoted to Practical Ophthalmology and Otology, including their allies, Rhinology and Laryngology." In those days ophthalmology and otology flourished together, while rhinology and laryngology were often linked with diseases of the chest. It was not many years before the reputation of this modest Journal spread from the "Western and Southern states" to the wide world, which has continued to give it the hoped for "substantial support."

The first issue comprised 74 pages. The initial Board of Editors headed by Doctor Parker, then Secretary of the Kansas City Polyclinic Post-Graduate Medical School, consisted of Doctor B. E. Fryer of Kansas City, Doctor Casey A. Wood and Doctor T. Melville Hardie of Chicago, and Doctor M. D. Lederman of New York City. The first editorial page announced that "original contributions are invited from all parts of the world. Those accepted, upon publication, will be liberally paid for. *It is to be understood that such articles are contributed exclusively to this Journal.*"

The first article was by D. B. St. John Roosa, M.D., LL.D., Professor of Diseases of the Eye and Ear, New York Post-Graduate



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A JOURNAL OF PRACTICAL OPHTHALMOLOGY, OTOLOGY, LARYNGOLOGY, AND RHINOLOGY.

EDITED BY

JAMES P. PARKER, M.D.

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Medical School and Hospital—Surgeon to the Manhattan Eye and Ear Hospital, and was entitled "The Best Operation for the Extraction of Cataract."

There were in this first number five articles directed to otolaryngologists:

"Excision of the Membrana Tympani and Necrotic Malleus in Four Cases of Chronic Suppurative Otitis Media." By Charles H. Burnett, A.M. M.D. of Philadelphia.

"Deafness the Result of the Poison of Syphilis; Its Treatment by Muriate of Pilocarpine in Acute Syphilitic and Labyrinthine Disease of the Internal Ear; Also Its Use in Chronic Disease of the Ear with Labyrinthine Symptoms." By Lawrence Turnbull, M.D. Ph. G. of Philadelphia.

"Otacoustic Treatment in Chronic Deafness." By J. A. Maloney, M.D. of Washington, D. C.

"Traumatic Hemorrhage of the Tympanum Causing Deafness with Subsequent Restoration of Hearing with Report of Cases." By S. MacCuen Smith, M.D. of Philadelphia.

"The New and Simple Method to Lessen Reaction After the Use of the Galvano-Cautery for Nasal Hypertrophies." By J. W. Gleitsmann, M.D. of New York.

At the very beginning the tail appears to have begun wagging the dog. Pages 210 and 211 tell the story. On the one is printed "*Special Notice—Removal. The* ANNALS OF OPHTHALMOLOGY AND OTOLOGY *has been REMOVED* from Kansas City to *ST. LOUIS, MO. . . . Assurance is given that the ANNALS will continually be chosen by the leading Specialists on both sides of the Atlantic for the presentation of meritorious contributions.*" Already the "Western and Southern states" were giving way to "both sides of the Atlantic," and the price mounted to $3.00. We read no more offers to pay liberally for accepted material.

The notice on the opposite page completes the picture:

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Kansas City, Mo.

At the end of the first year Parker wrote editorially "We are not a little proud to congratulate both ourselves and the readers that the Annals of Ophthalmology and Otology can no longer be

considered an experiment. . . . Our readers know by experience that they have been little worried by the verbose and useless inflictions of editorial vanity and appeal. Instead of this we have trusted in the strength of facts, believing that to 'do the deed and say naught of it' is the proper way to gain the confidence of discerning medical men.

"From the first we have relied upon no adventitious aids or outside business to establish and maintain our position, but we have resolutely held fast to purely professional and scientific ideals and interests."

But now things began to get really out of hand. The first volume had 212 pages, the second doubled this as Parker moved to St. Louis. By Volume 4 there were some 640 pages. Defensively Parker wrote, "The number of good original papers offered to the Annals continues to increase. . . . When two or more original articles upon the same subject are received, the shortest, most pithy and concise will take precedence." But it was no good. Volume 5 (1896) showed up with 1380 pages. The publication was now split into four journals: The Annals of Ophthalmology, The Annals of Otology, The Annals of Neurology and the Annals of Rhinology and Laryngology. The whole was under the management of a staff of ten and the rhinology and laryngology fell to H. W. Loeb.

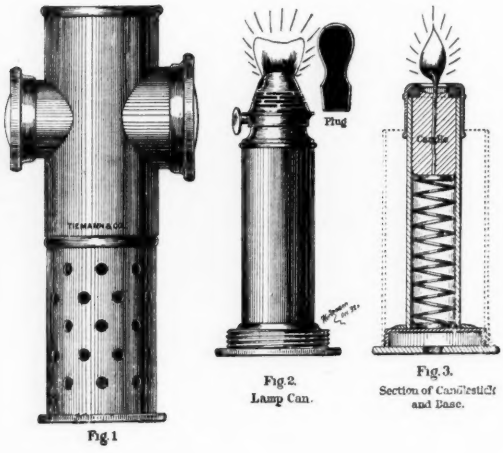
1897 brought Volume 6, reduced once more to portability and bearing its present title, THE ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY. It was now published by a brother of the founder and edited by a board of seven.

Among the contributors to Volume 1 we find Edward Jackson, then of Philadelphia, and Thomas J. Harris of New York, both still active in their specialties, still to be seen at meetings and both still taking a prominent part in the direction of educational thought.

In Volume 6 (1897), the first under the new name, appear Thomas Hubbard and J. F. Barnhill. Hubbard wrote on a topic not entirely ignored by present writers: "Angio-Neurotic Edema and Certain Vaso-Motor and Trophic Disturbances of the Mucous Membrane of the Respiratory Tract." In this volume there were only 25 original communications and hundreds of abstracts from the literature. Of the 25 original communications, 10 were case reports and descriptions of new instruments. There appeared also the Proceedings of the American Laryngological Association at

Washington, May 4, 5 and 6, 1897, the Proceedings of the American Laryngological, Rhinological and Otological Society, held in the same city, and the Proceedings of the French Otological Society, reported by J. Holinger, M.D. of Chicago.

At the close of the century the subject matter under discussion differed not very materially from that which occupies the journals of the present time, with a few notable exceptions. One of these was diphtheria. The index to Volume 7 devotes a special section comprising 34 titles to the disease. In this volume there are also four accounts of foreign bodies impacted in the esophagus, three of them requiring esophagotomy for their removal. There is described an instrument consisting of "a twisted stem or rod joined to a flattened handle together about 15 inches long. One end of the rod



The first illustration, from Volume I. A portable bedside lamp.

is bifurcated three inches, forming a closed loop. This loop is bent upon itself into two parallel loops an inch long and about half an inch apart, the part joining the parallel loops forming the beak of the instrument, which is placed at an angle of 45 degrees to the plane of the posterior parts of the loops and one-quarter of an inch from this plane.

"The coin or similar shaped foreign body is caught by the beak, deflecting into the parallel loops, and upon withdrawing the instrument the coin is drawn up with it".

In 1900 there was an article on the use of the new suprarenal extract in the nose. Its preparation, chemical and physiological properties were described and it was concluded that "the suprarenal extract is a powerful astringent without objectionable properties".

A case of acute thyroiditis of rheumatic origin was described.

The war between the external and internal sinus operators had already begun. Many an instrument was presented for the sewing, crushing and splinting of deflected septa and Doctor J. L. Goodale submitted some of the now endemic before-and-after photographs of patients with nasal deformities, but the most arresting title is undoubtedly "The Surgical Treatment of Otic Sclerosis by Doctor Ricardo Botey of Barcelona, translated by Albert Miller of St. Louis". The approach was endaural and the decompression consisted in the "mobilization of the stirrup after cutting adhesions to the border of the fenestra ovalis". Sometimes the stirrup was removed. Specimens and animal experiments were described. There are ten conclusions, the final one: "Since it is almost certain that otic sclerosis is a tropho-neurosis, with formation of connective and osseous tissue, principally in the labyrinthine capsule, round about the fenestra ovalis, in the cochlear canal and even in the helices of the cochlea, the neuro-epithelial terminations of the auditory nerve being more or less involved, surgical treatment of sclerosis of the ear is in nearly all cases useless".

1900 marked the accession to the editorship of Hanau W. Loeb of St. Louis, who was to retain the position until 1928 and to whom the ownership of the ANNALS finally passed. Through his guidance the Journal enjoyed a vigorous youth and a robust adolescence and his name became inseparably linked with it, so that it was known to most of his friends as 'Loeb's Journal'.

To those who knew Doctor Loeb it was patent that the quality of the early volumes would be maintained or improved. He was always very jealous of the standing of his publication and chose his material with great care.

While there is little in the early volumes which might be considered obsolete at this time, there is a notable absence of certain topics with which we are now familiar. Radiology is rarely mentioned and then only in connection with foreign bodies. There was no arsphenamine, no lipiodol and no vitamin. Allergy was met with only in its earlier guises of vasomotor rhinitis and asthma.

Some famous names appear: Jonathan Wright on Empyema of the Sphenoid Sinus with Delirium, Johann Sendziak on Laryngeal

Disturbances in Tabes, Samuel Allen on Lupus, LaFayette Page on Disease of the Upper Air Passage Relative to Mental Development, Alexander Randall on the Recording of Ear Cases, Joseph Beck on Pharyngeal Hyperkeratosis and George W. Crile on Operations for Malignant Disease.

Number 4 of Volume 11 (1902) contains the papers of the Sections of Laryngology and Otology of the British Medical Association with Gruenwald, Lack, Dundas Grant, Rohrer and Watson among the contributors.

In 1902 a few half-tones began to appear of a quality not above criticism, but in the following year small sections of the book were printed on glazed paper and the illustrations improved. In those days, as today, it was the particular concern of the publisher to select long-lived paper for his journal and even the early numbers are still free from discoloration.

While in the early volumes there are copious references to the transactions of the major special societies here and abroad there is none to an American otolaryngological journal prior to the Laryngoscope, which appeared in 1896. Presumably therefore the ANNALS was the first otolaryngological journal in America.

Under Loeb's aegis the ANNALS flourished and made many friends. Considerable numbers of its present subscribers have been on the rolls since the early days.

In 1927 Doctor Loeb died. The distinguished contributors to the Memorial Issue published in his honor the following year testify to the high regard which he and his journal enjoyed.

With a deep sense of obligation both to Doctor Loeb and to American laryngology, the editorship was assumed by L. W. Dean and Arthur Proetz, although the ownership of the Journal continued in Doctor Loeb's family where it remains today.

To mark the fiftieth year in the life of the ANNALS and to express in a fitting and concrete manner their acknowledgment of the debt which it owes to its many friends and contributors, the present editors have prepared this Jubilee Volume. The cordial response with which their invitation to participate in it has been greeted by the distinguished otolaryngologists of the country justifies the high hopes which the editors and managers hold for the future of the ANNALS.

III

RECOGNITION, TREATMENT AND PREVENTION OF HEARING IMPAIRMENT IN CHILDREN*

S. J. Crowe, M.D.

AND

Curtis F. Burnam, M.D.

Baltimore

It is taught in text books and believed by otologists and neurologists alike, that "impaired hearing for high tones with good hearing for low tones" is evidence of an inner ear lesion. Some examples of this type of deafness are shown in Figs. 2 to 7. We have accumulated abundant evidence in the Otological Laboratory of the Johns Hopkins Hospital that this type of hearing impairment may also be due to a middle ear lesion, which can be cured if recognized early.

We have been studying intensively for 16 years the causes, the pathology and possible methods of treatment for the prevention of deafness. For the first 14 years we used the method that has been employed so successfully in studying diseases of the heart, lungs, kidney and other parts of the body, i.e., the correlation of clinical examinations and functional tests with the gross and histologic appearance of the organ after death. This method has its shortcomings when a special sense organ like the ear is being studied. We have learned much about the pathology and the interpretation of hearing tests but very little about where the lesion began, the early symtoms, the primary cause of the trouble, how it progressed, how rapid was the progression and other facts that are essential before effective methods of treatment or prevention can be devised.

During the past 16 years we have made, for research purposes, audiometer, tuning fork and voice tests on more than 15,000 patients, approximately 3,000 of whom were children. These tests were all made in a sound-proof room with the 1-A Western Electric audiometer with a range from 32 to 16,384 d.v.

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*From the Otological Research Laboratory of the Johns Hopkins University and Hospital. Aided by a grant from the John and Mary R. Markle Foundation.

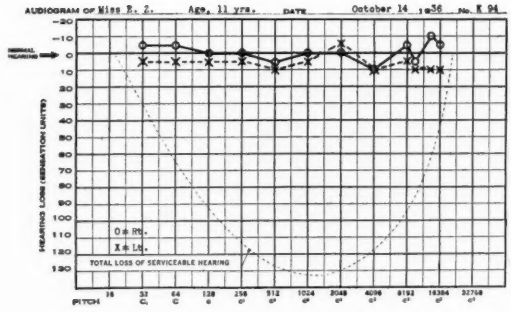
AUDIOGRAM OF Miss R. Z. Age, 11 yrs. DATE October 14 , 1936 No. R 94
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
O = Rt.
X = Lt.
TOTAL LOSS OF SERVICEABLE HEARING
PITCH

Fig. 1. Audiogram shows normal hearing in a child. Impaired hearing for high tones is often an evidence of inner ear or nerve lesion as shown in Figs. 2 to 7.

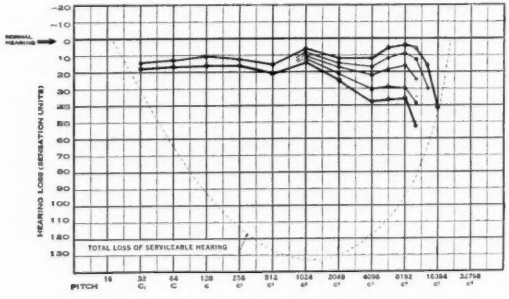
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
BUNCH and RAIFORD'S CHART IO TRANSFERRED TO STANDARD AUDIOGRAM FORM.

Fig. 2. Audiogram shows the age changes in hearing. The low tones change but little, but with each decade after the age of 30 years there is a progressive impairment for tones above 1024 d.v. (From Bunch & Raiford: Race and Sex Variations in Auditory Acuity. Arch. of Otolaryngology, Vol. 13, pp. 423-434, 1931).

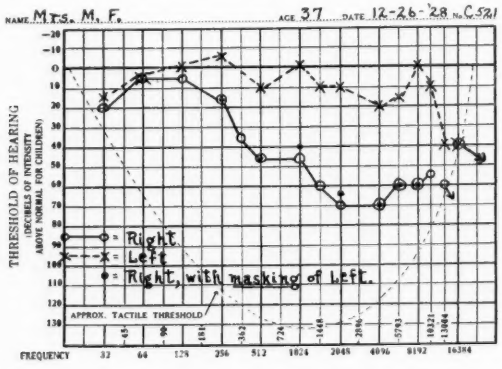
NAME Mrs. M. F. AGE 37 DATE 12-26-28 No. C 521
THRESHOLD OF HEARING
= Right.
= Left
= Right, with masking of Left.
APPROX. TACTILE THRESHOLD
FREQUENCY

Fig. 3. Audiogram. Acoustic tumor right, confirmed at operation. The pressure of the growing tumor on the cochlear nerve in internal canal causes impairment for high tones as the earliest auditory symptom.

As the years passed we have become more and more convinced that many of the hearing disorders of adult life begin in childhood, and if we are to learn more about the causes, treatment and prevention of deafness we must concentrate our studies on children.

From the studies made so far, we have learned that adenoids recur in more than 75 per cent of the children whose tonsils and adenoids have been removed before the age of puberty, and that the recurrence often interferes with the ventilating function of the eustachian tubes and leads to an insidious, painless type of progressive deafness. The explanation for the recurrence of lymphoid nodules in the oro- and nasopharynx is that lymphoid tissue is an integral part of the mucous membrane in this area, and it is impossible to remove all of it at operation. It is not the size, but the location of the lymphoid tissue that impairs the hearing. The appearance of the nasopharynx of these children (when examined with a nasopharyngoscope) is often identical with the "granular pharyngitis" so commonly seen in the oropharynx. In some the growth of lymphoid tissue is excessive, in others it is very slight. If we understood better the basic problem of why some children are more susceptible to respiratory infections, if we knew what is the relation between allergy, dietary deficiencies, ductless gland disorders and lymphoid hyperplasia, it would probably not be necessary to operate, radiate or treat the symptoms of lymphoid hyperplasia in any way. The cause could be corrected at the source.

We have also learned that almost every type of middle and inner ear deafness seen in adults is found in children from eight to fourteen years of age. There is this important difference, however: in children the impairment of hearing is just beginning and better opportunities are offered for study of the life history of the disease and its complications and for instituting some kind of effective or preventive therapy. In most adults the malady has been in the making for many years, and the hearing apparatus is irreparably damaged. There is so little chance that we will ever be able to cure deafness in the adult that we must seek for a clear understanding of the causes and methods of prevention in children.

In childhood, when the hearing impairment is just beginning, all types of middle and inner ear lesions, with the possible exception of otosclerosis, have one symptom in common—i.e., the hearing is more impaired for high than for low tones. This finding does not always indicate an inner ear or nerve lesion, even if the hearing by bone conduction is impaired.

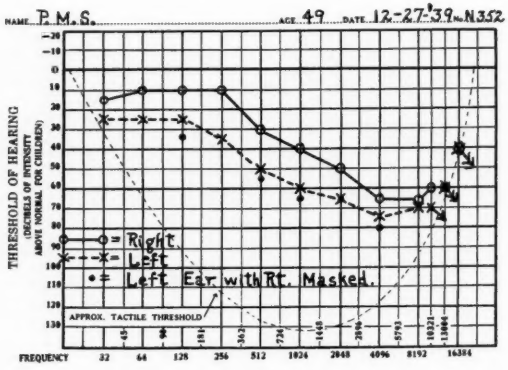

Fig. 4.

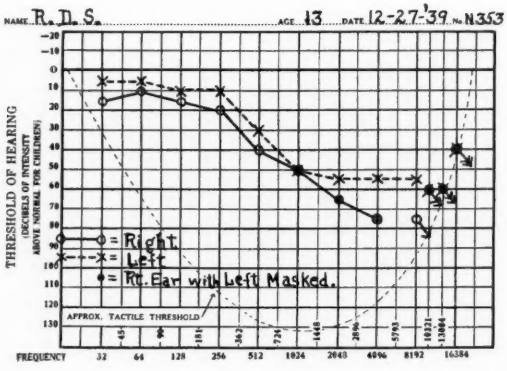

Fig. 5.

Figs. 4 and 5. Audiograms. Example of Manasse's hereditary nerve deafness in father and son.

We know that an overgrowth of lymphoid tissue, which partially obstructs the eustachian tubes for a period of years, causes changes in the mucous membrane of the middle ear. At first it is hyperemic, then passes into the secretory stage, gradually becomes myxomatous and finally fibrous tissue is formed, which interferes more and more with the mobility of the ossicles. These changes are very prone to occur in the attic, and on otoscopic examination the first evidence to be seen in the tympanic membrane is usually retraction of Shrapnell's membrane.

Proof that deafness of the inner ear type in children may be due to a long continued partial obstruction of the eustachian tubes with lymphoid tissue, is the demonstration that the hearing may be restored after radiation of the nasopharynx. Examples are shown in Figs. 8 to 10. We have followed many of these children for ten years or more, giving them repeated examinations with the nasopharyngoscope, the audiometer and tuning forks, and so determining the necessity for additional treatment. Some of these cases were reported by Crowe and Guild in 1938 and by Crowe and Baylor in 1939. We are convinced that approximately 50 per cent of the deafness in adult life can be prevented if school children are examined with this object in view. Both the hearing tests and the examinations must be directed toward the recognition of the first signs of impaired hearing and of conditions that may cause ear trouble.

The two common causes of deafness that can be prevented are: suppurations in the middle ear, which are usually secondary to infected lymphoid tissue; and long continued partial obstruction of the eustachian tubes by hypertrophic lymphoid tissue in the nasopharynx.

The function of the eustachian tube is to ventilate the middle ear, the pneumatized spaces in the mastoid and petrous part of the temporal bone, and the peritubal cells. If the eustachian tube is partially or intermittently occluded, the air in the middle ear and accessory cells is absorbed, the mucous membrane becomes congested and mucus or serum is poured out to fill the space. Complete blockage of the tubes causes subjective symptoms, but with partial or intermittent occlusion in children there is usually no tinnitus, no pain or other subjective complaints. The objective signs and symptoms, however, are:

1) Impaired hearing for the three or four highest tones (8, 10, 13 and 16,000 d.v.). These tones are far above the speech range, which lies between 250 and 3500 d.v., but as the changes in the

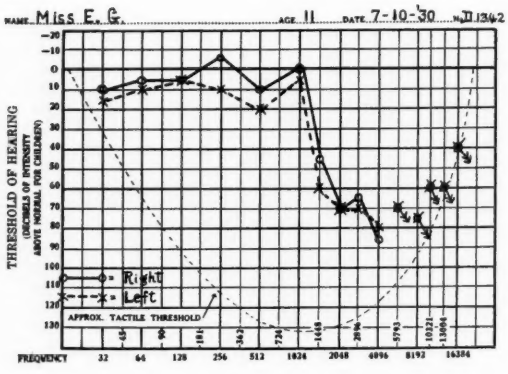

Fig. 6. Audiogram. This child hears no tones above 4096 d.v., while all tones below 1024 are well heard. No cause for the impairment was found.

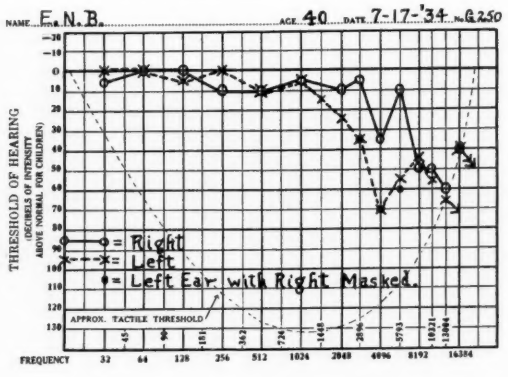

Fig. 7. Audiogram. Typical example of hearing loss due to detonation, in this case a shotgun near the left ear. Impaired hearing for high tones may also be due to a middle ear lesion as shown in Figs. 8 to 10.

mucous membrane of the middle ear progress and the movements of the ossicular chain become more and more impaired, the tones in the speech range gradually become involved.

2) Hypertrophic lymphoid tissue in the nasopharynx which grows in and around the pharyngeal orifice of the eustachian tubes causes, in addition to mechanical blockage and congestion of the mucous membrane in the middle ear and tube, a hypersecretion of mucus and eventually a myxomatous mucous membrane or fibrosis in the middle ear that interferes with the mobility of the ossicles.

3) Retraction of Shrapnell's membrane alone, or of the entire tympanic membrane.

4) Later the tympanic membrane often becomes so thickened and opaque that the middle ear mucosa cannot be seen with an otoscope.

These are the signs of impending ear trouble in children. If recognized early and properly treated the hearing may return to normal, or at least the progress of the deafness may be arrested. Temporary eustachian tube obstruction rarely impairs the hearing for more than a few days, but oft repeated or long continued partial occlusion causes fibrotic changes in the middle ear mucosa that permanently impair the mobility of the ossicular chain. When this has happened, no treatment will restore the hearing.

With the cooperation of the Baltimore City Department of Health and Board of Education, and with the support of the Markle Foundation and several interested individuals, we have examined during the past year 1365 children from public and parochial schools of Baltimore. They have all been between the ages of eight and fourteen years. The object of the examinations was to determine the incidence of impaired hearing. All but a small percentage of these children were thought by their teachers and parents to have good hearing, the defective children having already been placed in special groups or vocational classes.

We find: 1) that only 58.8 per cent of these 1365 children have normal hearing for all tones from 32 to 16,384 d.v. All tests were made in a sound-proof room (Fig. 11).

2) 36.3 per cent of these 1365 children have impaired hearing for high tones with normal hearing for low tones and the voice. It is a definite but a curious fact that this type of impaired hearing is much more common in boys than in girls. Only 30 per cent of

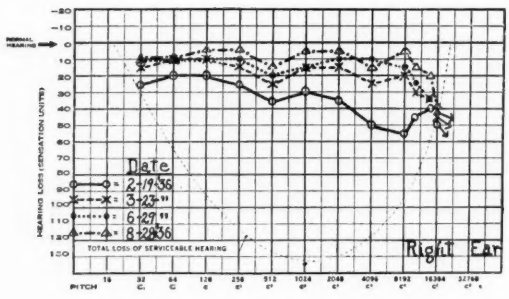

Fig. 8. The improvement followed radiation of adenoid tissue that interfered with the function of the eustachian tubes. This and numerous similar cases (in children) prove that middle ear lesions often cause more impairment for high than for low tones.

the girls were affected, but 52.7 per cent of the boys had impaired hearing for high tones with normal hearing for low tones and for voice (Fig. 11).

3) Only 5 per cent of these 1365 children have impaired hearing for both high and low tones, due to chronic suppurative otitis media, otosclerosis, and so forth.

4) Seven-tenths (7/10) of the girls have good hearing for all tones but only about one-half (½) of the boys have good hearing for all tones (Fig. 11).

5) Approximately one-half (½) of the 1365 children had had their tonsils and adenoids removed at the various hospitals in Baltimore, but the incidence of impaired hearing for high tones was about the same in this group as in the children who had had no operation (Fig. 12). In both groups the incidence was greater in the winter than in the autumn. The reason for this is shown by the nasopharyngoscopic examinations. Even in the children whose tonsils had been cleanly removed and whose oropharynx looked normal, it was found that the lymphoid tissue in the nasopharynx had always recurred to a greater or less degree. This tendency to recurrence is greater in some children than in others, and greater in boys than in girls. Lymphoid nodules are widely distributed in the mucous membrane of the nasopharynx, and to remove all of it at operation the entire thickness of mucous membrane would have to be taken out, which is manifestly impossible. It is a normal process for lymphoid tissue to hypertrophy in response to the stimulus of a

respiratory tract infection. Our studies lead us to the conclusion that following the removal of tonsils and adenoids in children, latent nodules, which were not evident before, often begin to grow after the operation. They may not cause obstruction to nasal breathing, but due to their location they do cause secondary changes in the middle ear and eventually impair the hearing for high tones—the beginning of what may be a progressive deafness. We repeat again, in order to have good hearing the eustachian tubes must function normally.

Otologists and pediatricians must realize that adenoid tissue recurs after operation, and for the preservation of hearing the nasopharynx should be examined twice a year up to the age of puberty, just as the dentist makes periodic examinations for preservation of the teeth.

When examination discloses a condition that endangers the hearing the proper treatment is irradiation and not a second operation. It has been known for many years that lymphoid tissue is extremely sensitive to ray therapy, and doses so small that they will not injure mucous membrane, cartilage, bone or central nervous system will always shrink lymphoid tissue. This result is only temporary, however, and it may be necessary to give two or three treatments a year for several years. By this procedure it is possible to maintain open eustachian tubes and tide the child over a difficult period.

The success of treatment depends on several factors:

1) The hearing impairment must be recognized and treatment begun in the earliest stage. Occasionally we see spectacular improvement in children with extreme deafness, but this is rare. To get the best results the loss of high tones must be stopped before impaired hearing has become evident in the school room or at home.

2) The type amenable to treatment is secondary to changes in the middle ear mucosa, which impair the mobility of the osscular chain.

3) The ear cannot function normally if there is any interference with the ventilation of the middle ear. Therefore it behooves us to examine with the greatest care the tympanic membrane, nasal passages, and nasopharynx of every deaf child and, no matter what is the primary cause of the deafness, make every effort to keep the eustachian tubes open and functioning normally.

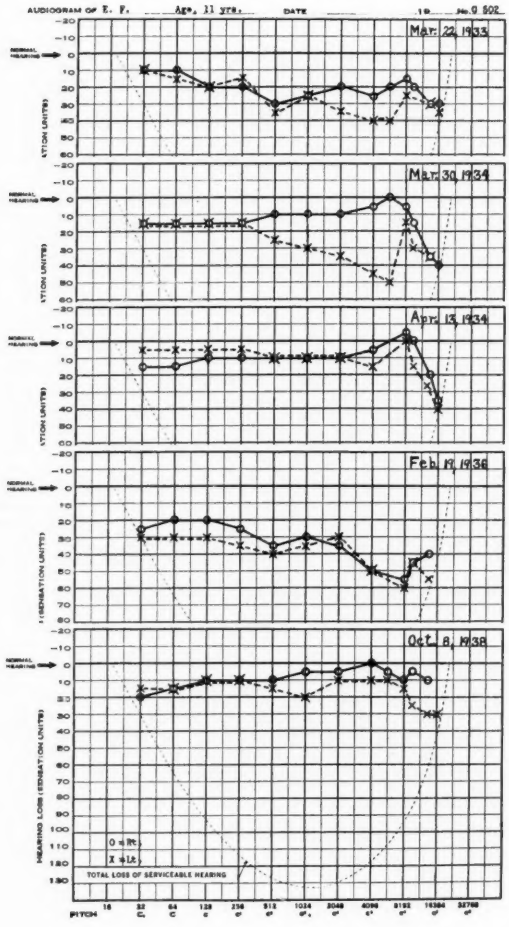

Fig. 9. Another case similar to that of Fig. 8.

4) It must be remembered that children have nearly all of the various types of deafness seen in adults and that all of these types have in their early stage one symptom in common—impaired hearing for high tones with good hearing for low tones. Therefore it is not possible to restore the hearing or stop the progress of deafness in every child with loss of hearing for high tones.

Successful treatment is accomplished by a variety of measures:

1) Removal of enlarged tonsils and adenoids followed by radiation to reduce lymphoid tissue in locations that cannot be reached by surgical methods, such as the nodules in the mucosa lining the tube, in the pharyngeal orifice of the tube and in the fossa of Rosenmueller.

2) Occasional inflation to remove thick tenacious mucus in the tube and middle ear. It may be advisable in some cases to do a paracentesis and with a catheter in the tube blow the mucus out into the external auditory canal.

3) General hygienic measures, diet, vitamins, change of climate, treatment of allergic tendencies, and, in short, everything that will improve the health of the child and lessen the frequency of colds.

Radiation treatment is an essential part of this program. In children, as well as in adults, it is desirable to have the method of application of radiation as simple as possible, a method that is as safe as possible, and one that can be repeated a number of times if necessary. At the same time it should be effectual in reducing or removing the adenoids and relieving the eustachian tube obstruction.

About 14 years ago, we came to a form of applicator very close to that which we are using at present (Fig. 13). This applicator is introduced through the nose and goes back directly against the nasopharyngeal posterior wall. It is then drawn over to one side or the other by means of a little adhesive strapping to the handle.

The handle is 13 cm. long. The tube into which the handle screws is 2 cm. long. The small glass tube containing radon is 1½ cm. long, the thickness of the tube wall is 1 mm. of brass, and the lumen is 1½ mm. in diameter.

About 50 mg. of pure anhydrous radium sulphate can be put into a tube of this size (2 cm. in length and 1½ mm. in diameter). Where radon is used, one can introduce any amount up to the equiv-

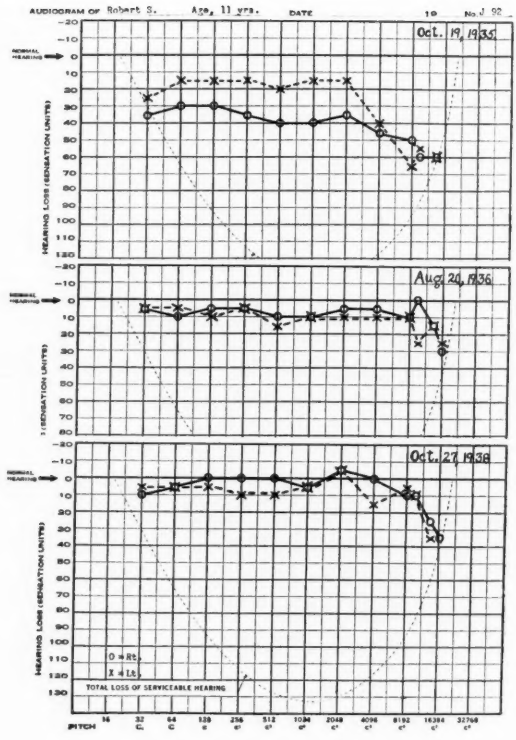
AUDIOGRAM OF Robert S. Age, 11 yrs. DATE 19 No. J 92
Oct. 19, 1935
Aug. 24, 1936
Oct. 27, 1938
HEARING LOSS (SENSATION UNITS)
O = Rt.
X = Lt.
TOTAL LOSS OF SERVICEABLE HEARING
PITCH

Fig. 10. Another case similar to that of Fig. 8.

alent of a gram or more of radium. Rarely, however, are such quantities indicated.

With this applicator, a dose of 2 gram minutes may be given to either side of the nasopharynx and repeated at intervals of four weeks or longer without causing irritation or disagreeable symptoms of any kind.

The duration of exposures will depend upon the amount of active material contained in the applicator. With 1000 mc. the time would be only 2 minutes on each side; with 100 mc., 20 minutes on each side; with 50 mg. or mc., 40 minutes on each side. The 1 mm. of brass filtration removes all alpha rays and a large portion of the beta rays. The actual mixture of rays emitted from the applicator is 95.5% gamma and 4.5% beta rays. The so-called secondary "K" radiation from brass is inconsequential.

The effectiveness of radiation on any tissue depends on the amount of absorption in the tissue. Rays which go directly through have no effect. In the mixture with which we are concerned, it is probable that the effects are about equally divided between the gamma and hard beta rays. This is due to the fact that all the beta rays are absorbed but only a small percentage of the gamma rays.

The quality of radiation which is emitted through the applicator walls is exactly the same whether radon or radium is employed. Radium has the advantage of being permanent and requiring no renewals. Its disadvantages are the relatively small amount of energy available in an applicator of this size, and dangers of loss by sterilization, by breakage, by theft, etc. The chief disadvantages of radon are the necessity of having a radon plant and the cost entailed in introducing the radon into glass tubes. In every other way it has real advantages.

It is a comparatively simple matter to radiate the nasopharynx with X-ray. The rays may be directed through the open mouth, but this method of approach is difficult even in adults, due to the time required for adequate treatment and the necessity for using a depressor to keep the tongue out of the field. For these reasons it is even more difficult in young children. If the rays are directed through the neck, the principle of cross-firing must be employed. Since considerably more than half of the rays delivered at the skin surface are absorbed by neck tissues before they reach the nasopharynx, it is evident that several portals must be used in order to prevent skin irritation. The fact that a large percentage is absorbed

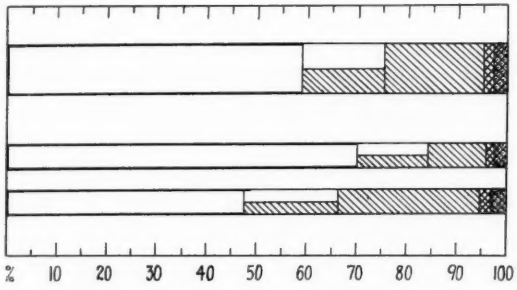

Fig. 11. Statistical studies on the incidence of impaired hearing in 1365 school children between the ages of 8 and 14 years. Only 58.8 per cent have normal hearing (unshaded zone above); 16.5 per cent have unilateral impairment for high tones; 19.8 per cent have bilateral impairment for high tones. In the remaining 4.9 per cent the hearing is impaired for other tones also. The sex difference in hearing is shown in the two lower strips: 70 per cent of the girls, but only 47.3 per cent of the boys have normal hearing for all tones.

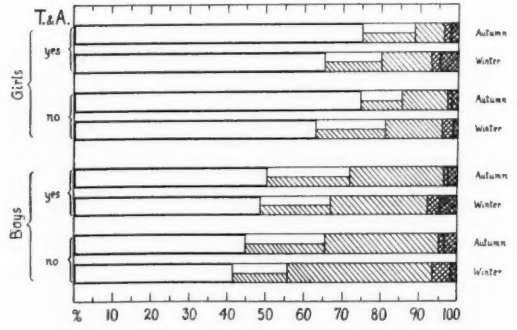

Fig. 12. Shows the influence of sex, the operative removal of tonsils and adenoids and the time of year on the hearing of school children. The highest percentage of normal hearing was found in the girls whose tonsils and adenoids had been removed and whose hearing tests were made in the autumn. The lowest percentage of normal hearing was in the boys whose tonsils and adenoids had not been removed and who were examined during the winter months.

necessitates a dosage on the skin surface much larger than is necessary to correct the lesion in the nasopharynx, for which the treatment is given. This excessive dosage subjects the centers of ossification for the growing bones of the face and skull to a large amount of radiation, which may do harm in a child and is of no value in the treatment of the primary condition in the nasopharynx. Furthermore it is difficult to maintain young children in a position for accurate cross-firing for the duration of the treatment. Successful results in shrinking lymphoid tissue in and around the orifice of the eustachian tubes and restoring the hearing cannot be expected unless the central beam in cross-firing actually passes through the area where treatment is most needed. Finally, X-ray treatments should not be carried out by anyone except an experienced technician or radiologist.

In contrast the radon nasal applicator can be used by the otolaryngologist who tests the hearing, examines the nasopharynx and places the applicator on the exact spot where the treatment is most needed. The applicator is small enough to pass along the floor of the nose of an infant. No anesthesia is necessary. With a strong applicator a three minute treatment is equivalent to fractional doses of X-ray that must be extended over a period of two or three weeks.

We wish to make it very clear that we do not advocate the indiscriminate use of radium as a treatment for deafness. It may or may not improve the hearing in a given case, depending on the cause and duration of the impairment. Radium is of great value in reestablishing the lumen and the function of a eustachian tube partially occluded by hyperplastic lymphoid tissue. In other words it is used to correct a condition that predisposes to impaired hearing and to attacks of otitis media. The best results are obtained when it is used in children with beginning impairment of hearing. Only rarely are spectacular results obtained in extremely deaf children or in adults.

In conclusion we wish to stress the following points:

1) "Impaired hearing for high tones with good hearing for low tones" is the earliest symptom of middle ear as well as inner ear deafness in children. We have abundant proof to support this statement.

2) The recurrence of adenoids after operation on young children is so common that it must be regarded as normal. This recurrent lymphoid tissue may impair the function of the eustachian

Fig. 13. The radon applicator is so small that it can be passed along the floor of the nose and into the nasopharynx of an infant. The capillary glass tube containing radon (sometimes as much as 1000 mc.) fits in the 1.5 mm. lumen of the brass tube, which in turn is screwed on the handle.

tubes and cause a low grade tubo-tympanic catarrh which may lead to chronic progressive deafness.

3) Recurrent or hyperplastic nodules of adenoid tissue in and around the pharyngeal orifice of the eustachian tubes are so located that they cannot be removed surgically.

4) Every type of deafness seen in adults is also found in children. In most types the earliest symptom is a loss for high tones, and for their early recognition the tones above 8000 d.v. must be tested.

5) In order to have good hearing the eustachian tubes must function normally. Impaired function of the eustachian tubes in children is caused by an overgrowth of lymphoid tissue in the nasopharynx. In order to see this condition the nasopharynx must be examined with a nasopharyngoscope.

6) Early recognition of impaired hearing for high tones—long before the hearing for speech is affected—and radiation of the nasopharynx to restore the patency of the eustachian tubes, is the only method for the prevention of progressive deafness of this type. If we are to learn more about the causes, treatment and prevention of deafness we must concentrate our studies more and more on children. The only hope is early recognition and treatment, before irreparable damage to the mucous membrane of the middle ear and ossicular chain has occurred. These changes begin in childhood. In the treatment of deafness, as in the treatment of cancer, the best results depend on early recognition, and in both the best results are the least spectacular.

7) Lymphoid tissue is so sensitive to radiation that the dosage employed in the treatment of the nasopharynx is far below the

amount that could cause any irritation or injury to the mucous membrane or surrounding structures. No scar tissue, no dryness of the nose or throat and no atrophic changes in the nasal mucosa follow treatment of the nasopharynx with radon, provided the proper dosage is given at the proper intervals. This statement is based on our observation of hundreds of children. Many of them have been followed for ten years or more. If used carelessly, or if the treatments are intrusted to a technician, radium therapy may be extremely dangerous. It should never be given in the presence of an acute nasopharyngitis. The object of the treatment is to reduce the size of lymphoid tissue. It has little or no direct action on bacteria.

Radiation is of no value in the treatment of chronic middle ear suppuration, otosclerosis or any form of inner ear or nerve deafness. It is used for the sole purpose of reducing obstructing nodules of lymphoid tissue, decreasing the secretion of mucus and restoring the normal ventilating function of the eustachian tubes. For this reason radiation is more often beneficial in children than in adults. To obtain the best results it must be used before changes in the middle and inner ear, secondary to partial tubal obstruction, have irreparably damaged the middle and inner ear structures.

JOHNS HOPKINS HOSPITAL.

IV

AN ANALYSIS OF THE OCULAR FUNDUS CHANGES IN OTITIC SINUS THROMBOSIS

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NEW YORK

Fundus changes have been described so frequently as a fairly common accompaniment of sinus thrombosis that it is necessary to refer to only a few observers. Ruttin in 1911 described 30 cases of sinus thrombosis among which were 8 cases of papilledema. Of these, 6 occurred after jugular ligation and operation on the sinus. The papilledema came on from two days to a month after operation. Dench in 1912 called attention to the fact that choked disc occurring during the course of suppurative disease in the temporal bone often signifies the presence of a sinus thrombosis. Atkins observed 40 cases of lateral sinus thrombosis in which eye-ground changes were noted in 17, 4 before and 13 after operation. Eves reported 38 cases with eye-ground changes in 10 per cent. Ginsberg reported 2 cases—in one a choked disc appeared a month after operation, persisted for six weeks and gradually receded. There was no evidence of increased intracranial pressure. The spinal fluid was normal, and there were no localizing signs. Tobey reported choked disc in 10 per cent of his cases. Maybaum and Goldman in a report of 9 cases of bulb thrombosis had 2 cases with papilledema, in both of which the swelling of the disc gradually receded after operation. White observed 33 cases with eye-ground changes—4 had dilatation of the veins; 13, blurring of the disc margins; 8, slight papilledema; and 8, marked papilledema.

In the 25 cases now being reported the fundus changes ranged from blurring of the disc margins to a measurable papilledema of as much as 6D. We have not included in this group instances with dilatation of the retinal veins per se. These 25 cases of fundus changes represent about 22 per cent of our cases of sinus thrombosis over the past ten years.

Although we are not convinced that operation on the lateral sinus or the jugular vein contributes to any considerable degree to the development of fundus changes, we have, nevertheless, set the

time of operation as an arbitrary barrier separating the two groups of cases, that is: (1) those cases showing fundus changes before operation on the sinus and jugular and (2) those cases showing fundus changes only after the sinus and jugular operation.

In Group I, there were 12 cases showing fundus changes before operation. Of these, 6 had blurring of the disc margins, 3 had optic neuritis, and the remaining 3 had choked disc varying from ½D to 6D. Of the 3 cases with choked discs, the choking was greater on the healthy side. In 11 of these 12 cases an interval of from one to three days occurred between the onset of fundus changes and the sinus and jugular operation. During this time there was no progress in the pathological changes in the eye-grounds. In the 12th case blurring of the margins was noted which in the three days before the sinus and jugular were operated, developed into a bilateral optic neuritis.

Of the 6 patients who had blurring of the discs before operation 2 went on to papilledema beginning three to four weeks after sinus and jugular operation. Two remained approximately stationary for two to four weeks and 2 developed peripapillary edema, which rapidly receded three days after operation.

Of the 3 patients who had optic neuritis before operation, 2 went on rapidly to papilledema within three to four days after the sinus and jugular operation, while the third promptly improved to almost normal within ten days. Of the 3 patients who had choked disc before operation, there was a slight increase in choking for about two to four weeks after the sinus and jugular operation, followed by recession. Of the 6 patients who had pre-operative blurring of the discs 4 had an obliterating thrombosis in the sigmoid sinus, and 2 had no demonstrable thrombus. Of the 3 who had choked disc before operation all had obliterating thrombosis.

The clinical courses up to recovery varied widely; in some the septic symptoms and the bacteremia continued for some time post-operatively, while in others the sepsis and bacteremia subsided almost immediately; in some convalescence set in soon, while in others the toxic symptoms with fever remained for many weeks. Yet there was no parallelism between the state of the fundi and the condition of the patient at any given time. In some the eye-ground changes were progressive after the patient was well enough to be out of bed, while in others there was an improvement in the fundi even though the patient was still quite sick. In all of these 12 cases the vision

remained good. In none of them did the optic nerve atrophy. In one there were vague signs of toxic encephalitis which subsided, and in no instance was there definite evidence of increased intracranial pressure. Of these 12 patients 11 recovered completely without mental or cerebral sequelae; one died of sepsis. It would seem that the abnormal fundus changes are in no way an index of the severity of the disease since but one of the 25 patients here reported died.

In Group II there were 13 cases showing normal fundi until after the sinus and jugular operation. These changes appeared with variable tempo ranging from blurring to optic neuritis and choked disc. In 5 of the 13 cases pathological changes in the fundi were noted within the first post-operative week; in 5 cases the changes appeared during the second post-operative week and in the remaining 3 cases the fundus changes appeared first on the 28th, 38th and 41st post-operative days. Of the 5 cases which showed abnormal fundi within the first post-operative week 3 began with blurring and did not materially progress, the process receding within two to four weeks. The other 2 began abruptly with papilledema and hemorrhages. One increased from 2D to 4D during the first post-operative month and then slowly receded; the other increased from 2D to 3D during the first week and by the end of the second week was receding. Of the 5 cases in which the pathological changes appeared during the second post-operative week the first case had blurring which did not increase but persisted for about three weeks; in the second case the blurring increased to optic neuritis and persisted, gradually receding to normal by the tenth post-operative week; in the third and fourth cases the blurring increased to a bilateral, low-grade papilledema of 1½D which receded completely in the ensuing three weeks; and in the fifth case blurring progressed to papilledema of 2½D in one eye and 1½D in the other with hemorrhages which persisted through eight post-operative weeks after which the patient was discharged. The remaining 3 cases in which pathological fundi first appeared on the 28th, 38th and 41st post-operative days are very instructive. The one appearing on the 28th day had a bilateral papilledema with hemorrhages, 3D in one eye and 2D in the other. On the 42nd day both eyes showed 2D. The regression in the fundi thereafter was very slow and extended over a few months. The one appearing on the 38th post-operative day also began with a bilateral papilledema with hemorrhages which rapidly increased to 2D, and here again the regression was spread over a few months. The one appearing on the 41st post-operative day began as a bilateral optic neuritis, and in a week there was a marked

recession. The operative findings in this group of 13 cases showing fundus pathology only after the sinus and jugular operation were as follows:

5 had obliterating thrombosis.

1 had a parietal thrombosis.

7 had no demonstrable thrombosis.

In this group of cases as in the first group with preoperative fundus changes the clinical course up to recovery varied widely; in some the symptoms of sepsis and the bacteremia continued, while in others the bacteremia and sepsis promptly subsided; in some, metastases occurred, while in others there were no metastatic foci; in some, convalescence set in soon, while in others the toxic symptoms with fever remained for many weeks. Yet here again, as in the former group there was no definite parallelism between the state of the fundi and the condition of the patient at any given time. In some the eye-ground changes were progressive after the patient was well enough to be at home, while in others there was improvement in the fundi even though the patient was still quite sick. In all of these cases the vision remained good. In none of them did the optic nerve atrophy. In two of these cases there were vague signs of toxic encephalitis which rapidly subsided. In no instance was there definite clinical evidence of increased intracranial pressure. Of all the cases none had the changes in the periphery and maculae so frequently seen in the fundi of patients with brain tumors.

It is interesting also that the clinical course with all the variable symptomatology and pathology described in this group of cases of sinus thrombosis showing fundus changes has its parallel in a similar group without fundus changes.

The modus operandi and precise mechanism of the fundus changes in sinus thrombosis is not known. It can be seen from eight of the cases reported where there was an obturating thrombus from the bulb to a short distance from the torcular that ligation of the jugular vein below the facial vein could not materially influence the intracranial venous return. This is also evidenced by the Tobey-Ayer test where pressure on the jugular on the side of an obliterating thrombus does not materially influence the intracranial pressure. In three of the cases described where the fundus changes first appeared weeks after the sinus and jugular operation, it is obvious that if ligation played an important part in the development of papilledema through stasis, then changes in the eye-grounds should have occur-

red soon after ligation, before the collateral circulation had an opportunity to supplement adequately the return flow. That the element of extended time in the formation of fundus changes can be disregarded is shown by the rapidity with which such changes can and do occur. A papilledema can develop in a normal fundus in a few days.

CONCLUSIONS

It must be obvious from the material studied and here reported that no firm basis as to the cause of papilledema in sinus thrombosis is established. If we discuss three causes which have been offered, (1) stasis, (2) increased intracranial pressure, (3) toxic state, we can find objections to all of them as causative factors. Dr. Dean, in a recent communication before the American Otological Society, stated that for 25 years he has observed the ocular fundi in every case of accidental opening of the sinus in which the sinus was packed in order to control hemorrhage. In none of these cases, he states, has he noted any evidence of papilledema from the blocking of the circulation in an uninfected sinus.

1. Stasis. Our reasons for the belief that ligation of the jugular does not, per se, contribute in any considerable degree to the development of fundus changes are the following:

a) In the reported cases, including our series, there were many in which papilledema occurred before operation.

b) In some, in which the fundus change occurred postoperatively, the papilledema came on four, five and six weeks after the stasis incident to ligation of the jugular must have spent its maximum effect.

c) In some instances there was operative evidence that the cranial venous channels of the diseased side were completely blocked. There was an obturating thrombus from the bulb to the torcular, and no bleeding could be established from either the inferior or the superior petrosal sinus. Under such circumstances, with the total abolition of the ipsolateral venous return flow, ligation of the jugular probably contributed little or nothing to the stasis.

2. Increased Intracranial Pressure. In the 25 cases reported, while not all had lumbar puncture, intracranial pressure was not evident clinically in any.

3. Toxic State. So far as a toxic factor is concerned, it is difficult to reconcile our findings with the belief that the toxic element

is an important etiological factor. As we have stated, in some cases the papilledema receded when the patient was quite ill and the fever and bacteremia continued, while in others the papilledema advanced when the patient was well enough to be out of bed and walking about.

The cranial sinuses are part of the dura, and the one factor which is common to all of these cases is, therefore, the inflammation of the dura. It is possible that the latter with its attendant edema and infiltration may be an important factor in the development of papilledema with sinus thrombosis.

36 E. 73RD STREET.

V

THE DIAGNOSTIC SIGNIFICANCE OF PARTIAL PARALYSIS OF THE FACIAL NERVE IN CHRONIC SUPPURATIVE OTITIS MEDIA AND MASTOIDITIS

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ROCHESTER, MINNESOTA

The otologists's interest in facial paralysis is centered in his need of being able to diagnose the site and nature of the lesion causing paralysis. In order to be able to make such a diagnosis the otologist must know the origin, course, branches, function and nerve connections of the facial nerve. In addition, he must be familiar with pathologic processes which may affect the nerve as it passes through the structures of the temporal bone.

GENERAL CONSIDERATIONS

Partial paralysis of the facial nerve invariably has been looked upon as being caused by an intracranial lesion which involves the nuclei. When such paralysis occurs in the absence of chronic suppurative otitis media, there is little doubt about the situation of the lesion. However, only a few references have been found in the literature to suggest that when partial paralysis of the facial nerve occurs in the presence of chronic suppurative otitis media, the lesion involves a certain portion of the nerve trunk within the temporal bone.

That facial paralysis has interested physicians and surgeons for a long time is evidenced by the fact that a large bibliography concerning the facial nerve has accumulated. Histologists and neural anatomists have contributed the results of their studies and research work so that it may be said that the knowledge of fundamentals is established. The surgeon's interest has been mainly to devise some surgical method to overcome facial paralysis so that disfigurement of the face may be relieved. Needless to say, disfigurement of facial expression caused by paralysis of the facial nerve has far reaching

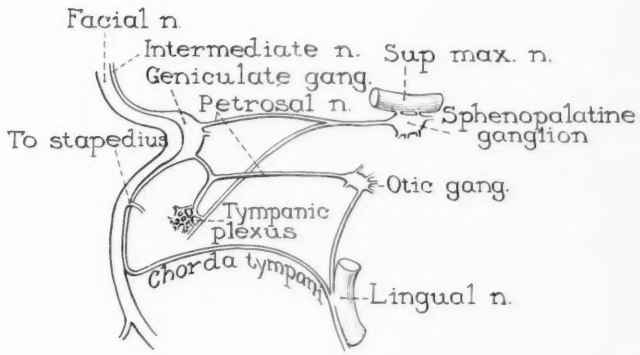

Fig. 1. Schematic representation of the connections of the facial nerve with other nerves and ganglia.

personal, social and economic implications. Therefore, any method of repair that will cause a return of function to the facial muscles is much worth while.

It would seem to be especially desirable that procedures be instituted in a given case before facial paralysis has become permanent, or before complete reaction of degeneration in the nerve fibers has taken place. Two types of surgical methods of repair have been devised. Much impetus recently has been given to repair of the nerve within the confines of the temporal bone, the usual site of injury in paralysis occurring before and after operations for disease of the mastoid process and middle ear. This method, for obvious reasons (because it utilizes the same nerve), seems entirely rational. Another method by which good results are achieved is utilization of the proximal end of one of the adjacent cranial nerves, for instance, joining of the spinal accessory or the hypoglossal nerve to the distal nerve trunk of the facial nerve as it emerges from the stylomastoid foramen. It does not fall within the scope of this report to mention all the observations that have led up to the present attitude toward surgical operations devised to overcome facial paralysis.

In 1937, Bunnell published an excellent article in which he brought the literature on the subject up to date and in which he reported the first successful operation for intratemporal repair of the facial nerve.

When I discussed with Prof. A. T. Rasmussen certain clinical observations that I had made during the past year which indicated that fibers in the nerve trunk held to a definite pattern of location, he said he did not know that such observations had been previously reported. In view of this statement on the part of such an authority as Professor Rasmussen, I felt that I should report my observations for what they are worth to others.

ANATOMIC CONSIDERATIONS

The seventh cranial nerve (the facial nerve) is a mixed nerve which has a motor root, the facial nerve proper, and also a sensory root, the nervus intermedius of Wrisberg. These two roots meet in the geniculate ganglion. The motor root arises from a nucleus situated mainly in the lower part of the pons, but some cells (namely, those for innervation of the orbicularis oculi muscle) originate as high as the nucleus of the third or oculomotor nerve, and other cells (namely, those for innervation of the orbicularis oris muscle) are situated as low as the hypoglossal nucleus in the medulla. In the pons the motor root of the facial nerve pursues a very tortuous course. It forms a loop which hooks around the nucleus of the sixth (abducent) nerve. As it leaves the ventral surface of the brain stem it enters the internal auditory meatus and passes through the temporal bone in the fallopian aqueduct. On the upper surface of the petrous pyramid within the fallopian aqueduct there is a swelling in the nerve which is called the geniculate ganglion. It is in the ganglion that the sensory root or the nerve of Wrisberg originates. The ganglion is connected with the sympathetic system by fibers from the otic ganglion. The geniculate ganglion is similar in structure to a posterior root ganglion and is entirely sensory in function. The cells of origin of the intermedius nerve form a very small, slender nerve trunk which can be identified in the internal auditory canal as it lies between the facial and auditory nerves. It runs centrally into the medulla along the fibers of the auditory nerve and joins the nucleus closely connected with the glossopharyngeal nucleus. Peripherally from the geniculate ganglion, fibers run along the great and small superficial petrosal nerves; others proceed along the trunk of the facial nerve in the chorda tympani. The cutaneous regions supplied by the geniculate ganglion, according to Ramsay Hunt's observations, include the tympanic membrane, the external auditory meatus and adjacent skin of the tragus, antitragus, concha and part of the lobule. The intermedius nerve conveys impulses of taste from the anterior two-thirds of the tongue upwards to the brain through the chorda tympani; this nerve terminates near the glossopharyngeal

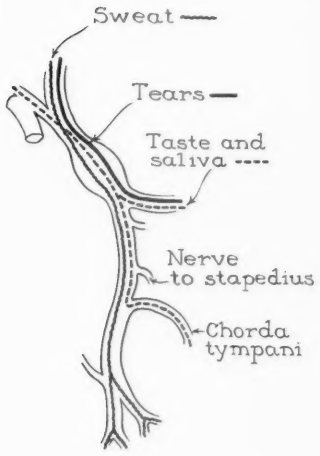

Fig. 2. Schematic depiction of fibers within the facial nerve which have functions other than motor.

nucleus. It also contains efferent secretory fibers which join the submaxillary ganglion and innervate the sublingual and submaxillary salivary glands.

Beyond the geniculate ganglion the facial nerve bends suddenly and proceeds downward in that portion of the facial canal which is situated in the upper part of the medial wall of the tympanic cavity and enters the middle part of the ear immediately above the oval window. From this point it runs in a backward direction under the anterior end of the horizontal vestibular canal and turns sharply downward behind the middle ear and leaves the skull at the stylomastoid foramen. As it passes through the middle ear it sends off a branch for the stapedius muscle, and the chorda tympani leaves the trunk of the nerve just beyond and passes through the glaserian fissure to join the lingual nerve. The communicating branches to the auricular branch of the vagus nerve are given off a short distance above the stylomastoid foramen. Reference to Figs. 1, 2 and 3 will show in a more graphic manner these relationships as outlined herein.

DIAGNOSTIC CONSIDERATIONS

Anatomic factors in diagnosis.—Diagnosis of the situation of a lesion causing facial paralysis may be determined accurately any-

where in the course of the pathway of the nerve from the lower end of the precentral cortical gyrus to the innervation of the peripheral muscle when disease of the middle ear is not present.[2] Cerebral lesions, because they cause the upper neuron type of paralysis, spare the occipitofrontalis, orbicularis palpebrarum and corrugator supercilii muscles. Lesions in the upper part of the pons would be accompanied by involvement of the pyramidal tract on the same side. A lesion in the lower part of the pons should produce involvement of the opposite side. If the lesion is in the medulla, the eye on the same side should turn inward from involvement of the sixth (abducent) nerve, and there should be accompanying effect on the pyramid and fillet.[3] Lesions proximal to the geniculate ganglion usually cause impairment of hearing and equilibrium. Involvement of the geniculate ganglion (Ramsay Hunt syndrome) is manifested by herpes of the external auditory canal and the adjacent auricle. If the lesion is situated distal to the geniculate ganglion, the resulting paralysis is of the lower neuron or flaccid type and involves all the facial muscles. All are familiar with the typical clinical picture of facial paralysis. If sensations of taste are absent from the anterior two-thirds portion of the tongue, the lesion is situated between the geniculate ganglion and the point at which the chorda tympani leaves the nerve trunk to pass through the middle ear. Involvement of the branch innervating the stapedius muscle or the chorda tympani is not always easy to determine in chronic suppurative otitis media.

It is apparent that for the patient to have the aforementioned syndromes, the entire trunk must be affected by the lesion. That complete involvement of the nerve trunk is not always present in peripheral lesions causing partial paralysis has been clinically observed.

Illustrative diagnostic points from the literature.—Randall recounted a case in which the face of a four months' old baby was partially paralyzed. The baby was found to have a tympanum filled with polyps. Mention was not made of the part of the face involved. In exploring to locate the origin of the polyps, Randall encountered a loose spicule of bone and removed it. It proved to be the anterior half of the stapes. The middle ear healed but the facial paralysis did not clear until after suppuration of the glands in front of the ear had ceased. Randall observed another patient who had slight facial paralysis. Paralysis increased to complete involvement in the course of a few weeks. Removal of a sequestrum at the apex of the cochlea was followed by recovery of facial function. In another case of a young woman, aged 21, "paresis of the lower distribution of the

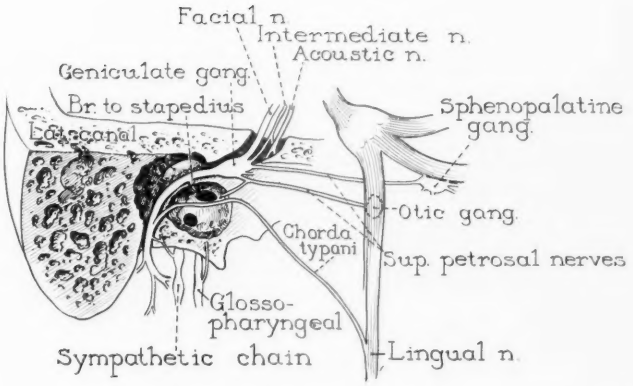

Fig. 3. Anatomic relationships of the facial nerve to its branches and important nerve connections as it passes through the temporal bone.

facial" on the first day after operation was observed. Full recovery from the facial paralysis did not take place until after a lapse of four years.

Weille,[6] in discussing a case reported by Cahill and associates at the meeting of the staff of the Massachusetts Eye and Ear Infirmary, said that because only the lower portion of the face was involved (in the case under consideration) he thought the lesion must have been centrally rather than peripherally located.

Pathologic factors in diagnosis.—Whether partial facial paralysis is encountered preoperatively or postoperatively in a given case, the portion of the face involved may provide the information necessary to determination of the site of the lesion.

Of most importance to otologists in evaluation of pathologic factors are the anatomic relationships of the facial nerve within the middle ear. The aural surgeon seems to be concerned with injury of the nerve below the level of the horizontal canal more often than he is with the promontory of the middle ear. Pathologic lesions are more likely to affect the facial nerve under the anterior end of the prominence of the horizontal canal and near the eminentia pyramidalis. Necrosis of the bone in this region or resorption of the bone caused by the insidious destrustive effect of cholesteatoma are the two main pathologic causes of involvement of the facial nerve.

AVOIDANCE OF TRAUMA AT OPERATION

During a radical operation on the mastoid process the facial nerve easily may be injured in the region in question in an effort assiduously to remove every vestige of disease. Great caution is necessary to avoid traumatization of the nerve with instruments. To help avoid such trauma the anesthetist must report any facial convulsive movement and be warned instantly to keep close watch of the face when the surgeon is working in this region.

REPORT OF CASES

CASE 1.—A woman, aged 45 years, had undergone a radical operation on the left ear for mastoiditis in November 1939. Twelve hours later it had been noticed that she was unable normally to draw in water through a drinking tube. The nurse in attendance had noticed that the lower portion of the face was paralyzed. Five days later paralysis of the face was complete. Galvanic and faradic stimulation of the muscles had been used for several months without improvement. The patient was admitted to the Mayo Clinic in March, 1940. At this time there was complete paralysis of the left facial muscles. The neurologic examination revealed a hysterical type of anesthesia over the distribution of the fifth cranial nerve which extended down to the clavicle. The patient's personality was much affected by the facial deformity. The cavity created by the aforementioned radical operation was well healed. In consultation with the neural surgeons, Drs. Adson and Baker, it was decided to perform an intratemporal bone exposure of the facial nerve after the method of Ballance and Duel. Exposure of the facial nerve from the region of the horizontal vestibular canal to the stylomastoid foramen revealed a defect in the outer aspect of the nerve at a distance of less than 1 cm. below the horizontal canal. It had been caused by impaction of a splinter of bone. The spicule was attached to a mass of inflamed tissue. The nerve had not been completely severed. The defect had filled in with a neuroma. Dr. Adson removed the neuroma and split the neurilemma of the nerve at this stage in the operation. Rapid healing of the wound took place. The patient was sent home and underwent treatment in the form of massage and electric stimulation. No improvement had taken place in six months. She returned in September 1940 and underwent spinofacial anastomosis, performed by Dr. Adson.

The salient feature in this case was that delayed postoperative partial facial paralysis followed twelve hours after operation. Within five days paralysis was complete. In radical operations for mastoiditis the chorda tympani usually is traumatized; consequently, testing for taste does not give particularly important information. In this instance, it may be assumed from the operative findings that injury to the outer aspect of the nerve caused paralysis of the lower half of the face. Later, swelling of the nerve within the facial canal caused complete paralysis. The patient insisted on spinofacial anastomosis because she was anxious to obtain return of function. In this respect it might be said that spinofacial anastomosis was instituted too soon after intratemporal decompression for me to know what the result

would have been if the spinofacial anastomosis had not been performed.

CASE 2.—A man, aged 37 years, presented himself complaining of pain in, and foul discharge from, the left ear. The ear had discharged intermittently since his childhood. The lower part of the face had been partially paralyzed for a week. It was the appearance of the facial deformity that had caused the patient to seek advice. He further stated that he had had attacks of vertigo for years. Surprisingly enough, this symptom did not concern him.

Examination of the left ear showed the canal to be filled with foul, purulent discharge. When the pus was removed, the middle ear was seen to be filled with polyps which bled on the slightest manipulation. Functional examination revealed that the patient heard a loud spoken voice in the left ear when the exclusion apparatus was used in the right ear. The labyrinth was not examined. A diagnosis was made of chronic suppurative otitis media, Type IV, with cholesteatoma and partial facial paralysis with probable circumscribed labyrinthitis. Operation was advised. Operation revealed that a cholesteatoma had produced a large cavity in the tympanic attic and antrum. On removal of the cholesteatoma it was seen that the horizontal canal had been completely eroded. The labyrinth was not disturbed further. The facial nerve had been exposed by the disease on the promontory above the anterior end of the horizontal canal. When the under surface of the nerve was manipulated, the muscles around the left eye twitched. All remnants of the matrix of the cholesteatoma were removed. On the third postoperative day the lower part of the face began to function. The patient was dismissed on the twenty-first postoperative day. Facial paralysis had completely disappeared, and hearing had improved to such an extent that the patient could detect a loud whispered voice in the left ear when the exclusion apparatus was used in the right ear.

It was the onset of facial paralysis that caused this patient to seek advice. He did not seem to be concerned about the seriousness of the condition of the ear. It interested me keenly to find again that it was involvement of the outer part of the nerve trunk that had caused paralysis of the lower part of the face and that touching of the under part of the nerve would cause the nonparalyzed facial muscles to move.

SUMMARY AND CONCLUSIONS

Clinical evidence is presented which seems to show that partial facial paralysis occurring before or soon after operations such as are mentioned herein has diagnostic significance in location of the part of the nerve trunk involved. The institution of early operative relief for the compressed or injured nerve seems to be a rational method of treatment. Partial facial paralysis occurring in the presence of chronic suppurative otitis does not necessarily mean that the lesion causing the paralysis is situated in the intracranial nuclei.

MAYO CLINIC.

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VI

THE RINNE TEST AND THE AUDIOMETER*

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The value of the Rinne test for differential diagnosis in clinical otology is quite generally accepted. Sonnenschein,[1] one of the foremost exponents of its use, expressed this opinion as follows: "Practical experience has shown that it is one of the most valuable of the tuning fork tests, and that it gives us a very good idea of the location of the affection, namely, whether it is in the middle or in the inner ear".

No attempt is made here to question its value. This study is concerned only with an attempt to compare the results secured by means of the Rinne test as ordinarily conducted with a tuning fork, and those secured by audiometric tests.

The usefulness of any new method of securing clinical information should be carefully compared with proven methods and should be considered with favor only if it reveals the same or additional information more accurately or conveniently or with less expenditure of time and energy. The Committee of the Section of Otology[2] of the Royal Society stated rather tersely in its report: "The results of testing by tuning forks have been for many years, and still are, the standard in otology, and until audiometers give results directly comparable with them, they cannot be accepted as a standard means of testing. New methods must be evolved from existing knowledge, and the Committee considers that future methods of testing must be based on the old-established tests by tuning forks".

The writer of this study in 1926 made these somewhat rash statements:[3] "There can be no doubt as to the value of the standard Rinne test. . . . So far as my experience extends, *there is no possible means of producing this test by an electrical device*". It now appears that such an audiometric test is available and that it is accurate as well as practical. The Rinne test is defined by Sonnen-

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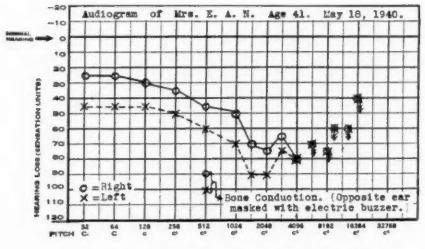

Audiogram of Mrs. E. A. N. Age 41. May 18, 1940.
O = Right
X = Left
Bone Conduction. (Opposite ear masked with electric buzzer.)
HEARING LOSS (DECREASE UNITS)
PITCH

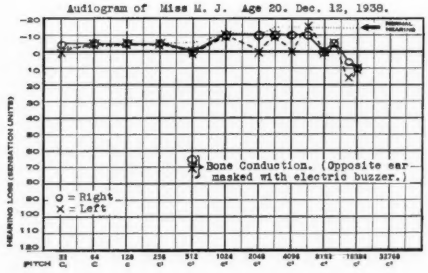
Tuning Fork Tests. (Weighted c^2 fork.)

Rinne { Right—Negative
Left—Negative.

Weber—Not Lateralized.

Schwabach { Right—Decreased 7 sec. (less than the normal).
Left—Decreased 10 sec. (less than the normal).

Fig. 1. The bone conduction threshold for the right ear at 512 d.v. is 90 db. and that for air conduction is 45 db., the difference between the two being 45 db. The corresponding threshold difference in the left ear is 40 db. Both ears gave the negative response in the Rinne test.


Audiogram of Miss M. J. Age 20. Dec. 12, 1938.
Bone Conduction. (Opposite ear masked with electric buzzer.)
O = Right
X = Left
HEARING LOSS (DECREASE UNITS)
PITCH

Tuning Fork Tests. (Weighted c^2 fork.)

Rinne { Right—Positive.
Left—Positive.

Weber—Not Lateralized.

Schwabach { Normal.
Normal.

Fig. 2. The difference between the bone conduction and air conduction thresholds is 65 db. in the right ear and 70 db. in the left.

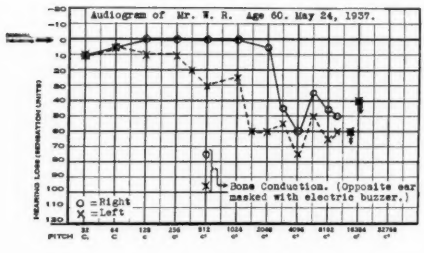
schein as a test "for the comparison of air conduction with bone conduction in the same individual". He should in addition have stated that this comparison should be for the same tone. Many writers within the last few years have been presenting audiograms which show the results of tests for both bone and air conduction. None, however, have attempted a comparison of bone conduction and air conduction to show the correlation between the audiometer records and the Rinne test.

It has been the custom for several years in the Otological Clinic of Washington University to do audiometer tests using the Western Electric 1A audiometer on all patients who enter with a complaint of hearing loss. In addition to the tests for the usual range of octave and semi-octave tones with the air-conduction receiver, the patient's ability to hear by bone conduction is also tested for the tone of 512 d.v., the Rinne, Weber and Schwabach tests are performed and, when it is deemed essential, tests for the spoken and whispered voice are done. If more accurate tests for spoken voice are necessary the Western Electric 4A phonographic test is used. All tests are done in a sound-proof room. A copy of a record of the tests, complete except for the voice tests, is shown in Fig. 1.

A weighted c^2 Bezold-Edelmann fork is used for the Weber, Rinne and Schwabach tests. A Western Electric receiver No. D80904 is used for the audiometer bone conduction test. The tone of 512 d.v. was selected for the bone conduction test because it, of the tones produced by the audiometer, most nearly corresponds in pitch with that of the weighted c^2 fork.

It will be noted from Fig. 1 that when bone conduction tests were done, the opposite ear was always masked. This is in accordance with the recommendations of the Committee on Bone Conduction Tests of the American Otological Society.[4] It has not been advised nor is it common practice, so masking was not done in the tuning fork tests.

In Fig. 1, the bone conduction threshold for the right ear at 512 d.v. is 90 db., and for air conduction the threshold is 45 db. The difference between the two threshold readings is 45 db. In the left ear, the threshold readings are 100 db. for bone conduction and 60 db. for air conduction for the same tone, the difference being 40 db. Both ears gave the negative response to the Rinne test with the c^2 fork. In so far as this particular case is concerned, it may be said that if there is a difference of 40 db. or 45 db. between the


Audiogram of Mr. W. R. Age 60. May 24, 1937.
HEARING LOSS (VIBRATION UNITS)
O = Right
X = Left
Bone Conduction. (Opposite ear masked with electric buzzer.)
PITCH

Tuning Fork Tests. (Weighted c^2 fork.)

Rinne { Right—Positive. / Left—Positive. } Weber—Not Lateralized.

Schwabach { Right—decreased 7 sec. (less than normal). / Left—Decreased 10 sec. (less than normal). }

Fig. 3. The bone conduction threshold for the right ear at 512 d.v. is 75 db. and that for air conduction is 0 db., the difference between the two being 75 db. The corresponding threshold difference in the left ear is 65 db. (95 — 30). Both ears gave the positive response in the Rinne test.

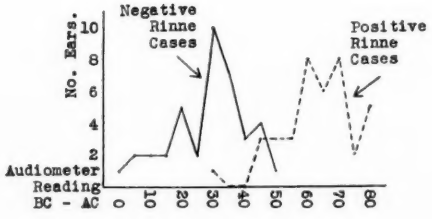


Fig. 4. Chart showing the distribution of the difference between the bone conduction and the air conduction thresholds (BC — AC) from audiometer tests of patients who gave the positive Rinne on one side and the negative on the other.

bone conduction and the air conduction thresholds, the Rinne test will be negative.

The record is Fig. 2 is submitted for similar analysis. The hearing in this case is essentially normal. There was no history of hearing loss, and the complete otological examination was negative. (It will be noted that the curve of normal hearing with this audiometer in the sound-proof room does not correspond exactly with the normal zero line of the chart. The line of dots shows this normal.) As in the case shown in Fig. 1, the bone conduction threshold for the right ear at 512 d.v. is 65 db. and that for air conduction is 0 db., the difference being 65 db. In the left ear, the corresponding difference between the bone conduction and air conduction thresholds is 70 db. Both ears gave the positive response in the Rinne test. The measurements in this case show that if a difference of 65 or 70 db. exists between the bone conduction and air conduction thresholds the Rinne test will give a positive response.

Figure 3 is the record of a soldier who began his service in the artillery in 1898. The tests give the classical picture of perceptive or nerve type deafness, bilateral, more marked in the left ear. The Schwabach and the audiometer tests both show decreased bone conduction; the Rinne was positive on each side, but as might be expected, the Weber was not lateralized. The patient attributed the hearing loss in his left ear to the effects of the noise of a shell explosion which he remembered distinctly. He was not aware of any particular loss in his right ear.

In the right ear the difference between the threshold readings for bone conduction and air conduction at 512 d.v. is 75 db. (75 db. — 0 db.), and in the left, the threshold difference is 65 db. (95 db. — 30 db.). One the basis of the tests in this case it may be said that if there are differences of 65 db. or 75 db. between the bone conduction and air conduction thresholds at 512 d.v., the Rinne will be positive.

The principle which has been illustrated in these three cases cannot be accepted practically unless it can be shown to hold in a large number of cases. Figure 4 shows graphically an attempt to demonstrate it in 39 consecutive cases which had the negative Rinne response on one side and the positive on the other. It will be seen that the difference between the bone conduction and air conduction thresholds in the ears which gave the negative Rinne ranged from 0 db. (in one case) to 50 db. (in one case); the greatest number (ten cases) had a threshold difference of 30 db. That this is not a

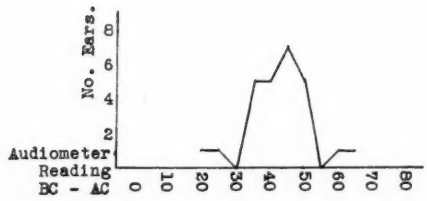


Fig. 5. Chart showing the distribution of the difference between the bone conduction and the air conduction thresholds (BC — AC) from audiometer tests of cases who gave the "neutral" Rinne response.

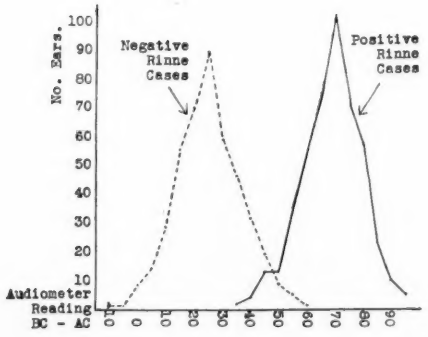


Fig. 6. Chart showing the distribution of the difference between the bone conduction and the air conduction thresholds (BC — AC) from audiometer tests of 216 patients who gave the negative response to the Rinne test in both ears and 232 patients who gave the positive response in both ears.

smooth curve of normal distribution is probably due to the fact that the number of cases is small.

The opposite ears in these cases which gave the positive Rinne response had thresholds ranging from 30 db. (in one case) to 80 db. (in five cases). The great majority had a threshold difference of 65 db. (plus or minus 5 db.).

Of the negative Rinne cases, only five had differences of 45 db. or more, and of the positive Rinne cases, only four had differences of 45 db. or less. It appears that the curves cross near 45 db., and that if any case had a threshold difference of 45 db. it might give either a negative or a positive response in the Rinne test.

The result of the Rinne test is at times difficult to determine in certain cases because they apparently hear the sound of the fork equally well by both bone and air conduction. The response in these cases has been variously called the "neutral", "indifferent" or "indeterminate" Rinne. Twenty-two cases of this type were available for study. The difference between the bone conduction and air conduction thresholds is shown graphically in Fig. 5. The range of threshold differences lies between 20 db. and 65 db., the great majority being between 35 db. and 50 db. on the graph. It will be noted that the peak of this curve is at 45 db., the point near which the curves of Fig. 4 cross. While the number of cases is small, it appears significant that this point in both curves so closely coincides. This should also be noted in connection with the curves shown in Fig. 6.

Cases with the positive Rinne or the negative Rinne response in both ears which were available for analysis were considerably greater in number than those used in Figs. 4 and 5. Record of 231 patients who gave the positive Rinne response and of 216 who gave the negative response in both ears were available for study and comparison. It is thought that groups of this number were large enough to establish this correlation more definitely. The results of the analysis of these are shown in Fig. 6. There is considerable variation in the threshold differences of each group, but the results appear to follow a curve of normal distribution. It will be noted that the threshold difference for the negative Rinne cases extends from —10 db. to 60 db., the peak being at 25 db. The results for the positive Rinne cases extends from 35 db. to 95 db. with the peak at 70 db. As in Fig. 4, the curves for the two types of response cross near 45 db. indicating that a few in each group might well be classified with those of Fig. 5 and called "neutral" cases.

SUMMARY AND CONCLUSIONS

When tests for both bone conduction and air conduction are done with the audiometer, opportunity is offered for making a comparison of these two threshold readings for any tone produced by the audiometer. A similar comparison of the results of such tests with the tuning fork is the basis for the standard Rinne test which is widely accepted in clinical otology. In this study, a comparison of the thresholds for air and bone conduction secured by tests with the audiometer for the tone 512 d.v. is made with the results secured in the Rinne test using the c^2 weighted tuning fork. It is shown in a great majority of cases that if there is a difference of more than 45 db. between the bone and air conduction thresholds as determined by the audiometer, the Rinne will be positive. If this threshold difference is less than 45 db., the Rinne will be negative. If this threshold difference is 45 db. (plus or minus 5 db.), the Rinne will be "indeterminate" or "neutral". The range of variation of these threshold differences offers opportunity for the study of its clinical significance, i.e., the significance of the so-called "shortened", "lengthened" or "normal" positive and negative Rinne responses.

At least two factors of error in the standard Rinne test can be eliminated by use of the audiometer for this test: (1) the error due to the constant decrement of the fork, for the intensity of the tone used with the audiometer for both bone and air conduction remains fixed for each setting of the audiometer intensity dial; (2) the error due to possible variations in pressure and in the selection of the point of contact against the mastoid process is eliminated because it is possible to maintain the tonal intensity without change while the pressure is varied at will or while the optimum point of contact is being located.

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VII

AN EVALUATION OF THE MÉNIÈRE SYNDROME

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Milwaukee

In 1861, almost seventy years ago, Ménière[1] described a group of symptoms which he believed constituted a separate disease entity. From the description of the pathology of one of his cases which came to autopsy, it would appear that in that particular case he was dealing with a purulent labyrinthitis. His original report of the symptomatology of the Ménière Syndrome was classic and left very little to be added by future clinicians.

He described the sudden attack of vertigo without any apparent preceding illness, the recurrence of the attacks, the extreme prostration of the patient and the tendency of the attack to recur. He noted that tinnitus preceded the first attack, persisted between attacks, was often increased during the attack and was not influenced by pressure upon the carotid. He noted the impaired hearing in one and sometimes in both ears, which began with the tinnitus and progressed to complete deafness without the evidence of any ear disease. In a series of papers he described nine such cases.

From the foregoing, it is clear that Ménière had a very clear conception of the syndrome which now bears his name, and that very little regarding the symptomatology of the condition has been added since his time.

ETIOLOGY AND PATHOLOGY

Ménière[1] felt that he had found the cause of the condition when he observed at postmortem a bloody fluid in the labyrinth and interpreted this as due to a labyrinthine hemorrhage. We now know that his view was incorrect, but we are still in the dark as to the etiology and as to the pathology of this condition although numerous views on these particulars have been expressed from time to time. Nor are we at all sure that the condition described by Ménière constitutes a clinical entity. The condition is not rare, and large series of cases have been studied by various observers (Mygind and Dederding,[2] Furstenberg,[3] Dandy,[4] Crowe,[5] Walsh and Adson,[6] and others).

The etiological factors described by various authors for the cause of the syndrome have been legion, but in the opinion of recent investigators the etiology is more or less definitely concerned with a lesion in the cochleo-vestibular apparatus, and inasmuch as the entire symptomatology concerns the cochleo-vestibular apparatus the lesion could affect the acoustic nerve, its peripheral distribution or its central connections.

Because of the fact that their patients presented not only aural symptoms but extra-aural as well (headache, vasomotor rhinitis, chilly sensation, rheumatism, gastro-intestinal disturbances and nervous phenomena, such as easy fatigability, irritability, depression, insomnia and loss of memory), and because the aural symptoms were, transiently at least, relieved by pilocarpine medication, and because they noticed that their patients became worse after an increase in weight and improved after a loss of weight, Mygind and Dederding[2] and D. Dederding[7] came to the conclusion that they were dealing with an abnormal water metabolism causing fluid retention in the labyrinth and other portions of the body from which symptoms were emanating; that this "extra cellular edema" in the labyrinth caused the aural symptoms; that in the meninges it produced the headache, in the mucous membranes the vasomotor rhinitis, and in the fascia of the joints and in the muscles the rheumatism.

Kopetsky[8] also believed that there must be some disturbance in water metabolism which gives rise to a waterlogged condition of the static labyrinth.

Furstenberg[9] believed it "doubtful if any of the clinical manifestations of Ménière's symptom complex can be definitely ascribed to any isolated part of the auditory tract" and thought that "any or all of its constituents may participate in the changes which give rise to a typical attack." He decided that Mygind and Dederding were in error in ascribing the etiology to a disturbed water metabolism for the reason that in eliminating large amounts of water by perspiration and diuresis Mygind and Dederding were also eliminating electrolyte. Furstenberg, therefore, came to the conclusion that the relief of symptoms in the cases of Dederding and Mygind was due to the elimination of sodium from the body. And in this connection it is recalled that Mygind and Dederding also put their patients on a low sodium diet, for the specific purpose, however, of increasing diuresis.

Dandy,[10] McKenzie,[11] Adson[6] and others have called attention to abnormal arterial loops springing from the internal auditory artery

or the inferior cerebellar artery, surrounding and in contact with the auditory nerve, and believe that these arterial loops may be a factor in the production of the symptoms in at least some of their cases. In this connection Torkildson[12] found at operation an abnormal artery about the size of the facial nerve passing into the internal auditory meatus along with the acoustic and facial nerves. Removal of the artery alone was followed by a remission of the syndrome for four months, i. e., up to the time of his publication.

If such arterial loops cause the symptoms of Ménière's disease they must operate by pressure upon the nerve, and Crowe,[5] Furstenberg[9] and Dandy[10] have all examined these nerves microscopically and found them normal. Furthermore, as Crowe points out, such arterial loops could damage the nerve only if they showed arteriosclerotic changes, and cerebral arteriosclerosis is extremely rare in the entire group of the Ménière Syndrome cases. He makes the further point that if these arterial loops were causing damage to the acoustic nerve within the confines of the internal auditory meatus, one should also expect damage to the facial nerve, which is never found in this condition.

That allergy plays some role in the etiology of the Ménière Syndrome is apparent from the reports of various observers showing that the Ménière Syndrome frequently appears in patients with other allergic manifestations, that the withdrawal of the offending allergens will cause a cessation of the attacks and that the readministration of the allergen will again produce the attack. (Dean, Kobrak, Dederding, Proetz, Vogel, Urbach and Wilder, Malone, Rowe, Duke, all cited by Dean,[13] Dohlman,[14] Grandstrom and Nylen.[15])

In fact, Vogel[16] found that of 83 patients with the Ménière Syndrome, 5 had a history of hay fever, 9 of urticaria, 15 of vasomotor rhinitis and 5 of asthma.

It is well known that a vasomotor instability and an increased capillary permeability are features of an allergic condition. If this is so, and if the majority of patients with the Ménière Syndrome are allergic, and if such an allergic reaction can alter the water metabolism and produce localized waterlogging of tissues or change the mineral metabolism with the same resulting localized edema, then the causation of the Ménière Syndrome could be logically explained on an allergic basis in a majority of the cases. The localization of the trouble is not in the nerve alone because no pathology has ever been found there microscopically. The pathology is not exclusively in the end organ because section of the acoustic nerve does not abolish tin-

nitus although it cures the vertigo. However, a bio-chemical process, whatever its cause, operating both in the end organ and also in the basal nuclei and producing "extra-cellular edema" in both regions, could adequately explain all the aural manifestations of the syndrome.

Crowe,[5] Dandy[10] and others have emphasized the fact that because of the lack of postmortem material the exact pathology of the condition, even in the end organ, is unknown. Basing his conclusions upon the microscopic examination of the temporal bones of two individuals who had died following an intracranial section of the vestibular nerve, Hallpike[17] presents an interesting hypothesis on the causation of the intermittent recurring attacks of vertigo and the progressive deafness of the Ménière Syndrome.

His sections show a gross dilatation of the entire endolymph system. Reissner's membrane was pushed against the walls of the cochlea and adherent thereto, thus obliterating the scala vestibuli. The tectorial membrane was flattened down on the organ of Corti, which showed marked degenerative changes. There were also degenerative changes in the sensory epithelium of the vestibule and of the stria vascularis. Whether this condition was due to an oversecretion of the endolymph or its faulty absorption he could not tell, but he assumed the latter to be true because an absence of perisaccular connective tissue was noted.

He explains the individual attacks of vertigo as being initiated by bouts of asphyxia of the labyrinthine end organs, brought about by extremely rapid rises of fluid pressure in response to relatively small volume increases of endolymph. But before this can happen the cochlear duct must first have dilated to the limits of its bony walls.

SYMPTOMATOLOGY AND DIAGNOSIS

The symptoms of the Ménière Syndrome are characteristic and well known. It is a condition of adult life, most of the cases beginning in the third, fourth and fifth decades. While the extremes of life are usually spared, Torkildson[12] reports a case at the age of three and Crowe[5] two cases aged six and fourteen respectively.

The condition usually begins with a sudden characteristic violent attack of vertigo as in 74 of Crowe's series of 119 cases. However, the first symptom may be deafness, usually unilateral and often of abrupt onset as in 14 of his cases, or tinnitus, as in 31 of his series.

The predominating and most disabling feature of the condition is the vertigo. This comes on in a sudden, violent apoplectiform attack so severe as to throw the patient to the floor or force him to clutch at anything for support. This vertigo is characterized by a sense of whirling in the patient himself or in the objects about him and is aggravated by any movement of the head. It may keep up for hours or even days and weeks. In the intervals between the attacks the patient feels quite well. When the condition first begins the characteristic attacks of vertigo occur at long intervals, but as time goes on they recur with greater frequency and severity so as to completely disable the individual. They are frequently associated with nausea and vomiting. As Dandy[10] has pointed out, the vertigo differs in its intensity from that produced by any other intracranial conditions such as acoustic neuroma, angle tumors, degenerative lesions and the like. Like the vertigo occasioned by a labyrinthitis in kind, degree and intensity, it differs from that vertigo in that once a labyrinth has been put out of commission by a labyrinthitis or by a pyramid fracture, the attack of violent vertigo runs its course in a few weeks, never to repeat itself.

Nystagmus may accompany the attack but does not always do so. McKenzie[11] and others are of the opinion that neither the directions of the nystagmus nor the direction of the vertigo have any lateralizing value. Dandy,[4] Crowe,[5] McKenzie,[11] Furstenberg[9] and Walsh and Adson[6] are all of the opinion that the caloric responses are of no value in making a diagnosis or in lateralizing the affected side.

The deafness usually follows the initial attack of vertigo, but may precede it (14% in Crowe's series). It is usually unilateral but may be bilateral (38% of the series of Walsh and Adson[6]). It is of the inner ear or perceptive type and is progressive.

There is nothing about the tinnitus to distinguish it from that arising from otosclerosis or any other middle ear lesions, as Crowe[5] points out. Mygind[2] thinks that the tinnitus is due to an intracellular edema in Corti's organ, but if this were so it should disappear in every case after section of the acoustic nerve, and it does not. The author would rather believe that it is due to a similar concomitant pathological process in the end organ and in the cochlear nuclei. Such a process, probably edema, in the cochlear nuclei would explain the persistence of the tinnitus after section of the eighth nerve.

In cases of bilateral deafness the presence of a unilateral tinnitus may be a very valuable lateralizing sign.

Headache is one of the frequent extra-aural symptoms of the Ménière Syndrome, and Dederding reported the presence of headache in 91 per cent of her series of 228 cases. Cohen[18] reviewed 126 cases and found headache present in 114 (91%), and the histamine treatment recently promulgated by Shelden and Horton[19] is the outgrowth of the use of this medication by one of them (Horton) in the treatment of vascular headache.

The attacks of the Ménière Syndrome are usually not preceded by any prodromal symptoms, but in a review of 100 case histories Crowe[5] found that there were aura in 13, increased tinnitus in 6, fullness of the head in 2, faintness and nausea in 2, increased deafness and tinnitus which improved after the attack in 1, burning sensation in the throat in 1, tingling and numbness of the fingers of both hands in 1. Preliminary aura have also been noted by Walsh and Adson[6] in a few cases.

Crowe[5] obtained a history of an occasional loss of consciousness in 15 of 117 patients. This was never accompanied by convulsive movements or any other evidence of epilepsy, and as the vestibular nerve was subsequently sectioned in these 15 patients and as the loss of consciousness never recurred, Crowe assumed that it was in some way connected with the vertigo and probably resulted from a sudden loss of blood pressure.

Spontaneous remissions of the vertigo and sometimes even of the tinnitus occur in this syndrome for no particular reason. Thus it is questionable whether one can ever speak of an actual cure of the condition. In Crowe's[5] series of 117 cases there were 48 who had such remissions lasting from a few months up to 12 years. As such remissions are also known in allergic states it strengthens my opinion that allergy plays some role in the production of this symptom complex.

In the average case the diagnosis of this condition should not be difficult. While there is nothing about the deafness to distinguish it from perceptive deafness due to other causes, and while the tinnitus can also result from other ear pathology, the apoplectiform attack of vertigo with a sense of rotation cannot be mistaken for that due to any other condition with the possible exception of a direct invasion of the labyrinth by trauma or disease, and from this it is differentiated by the fact that it recurs in attacks of gradually increasing frequency and severity. The dizziness caused by acoustic neuroma, angle tumors and other types of intracranial pathology is

never of the same severity and is usually produced or aggravated by movements of the head.

And that brings me to the discussion of a pseudo Ménière Syndrome. In the true Ménière symptom complex the attack of vertigo is usually the first symptom in a large majority of the cases. In some the tinnitus and deafness soon follow, in others these symptoms are delayed in their appearance for long periods of time, and in a few the tinnitus and deafness never occur. It is quite possible that these are all gradations of the same condition, and if the attack of vertigo is characteristically sudden and severe with a definite sense of rotation present and if it disappears only to recur again, I believe the condition should be referred to as a Ménière's Syndrome even in the absence of tinnitus and deafness.

On the other hand, we, as otologists, see many cases of unexplained and unexplainable tinnitus and deafness. It is also possible that some of these cases are way stations in the development of a true Ménière Syndrome and that if we observe them long enough the vertigo may appear in some of them.

TREATMENT

As would be expected because of the varying views of the etiology and pathology of this condition the treatment in the past has been almost as varied as the physician's conception of the symptom complex and has included most of the agents of physiotherapy and many of the drugs in the pharmacopeia. At the present time the most efficacious treatment of the condition may be divided into medical and surgical.

Having observed increase in the body fluids in cases of the Ménière's Syndrome and believing that this was due to an extracellular edema, secondary to deficient capillary function, Mygind and Dederding[2] and Dederding[7] believed that the aural as well as the extra-aural symptoms in the condition were due to a retention of water. They had noted that their patients became worse when they were overweight and improved when they lost weight. Their treatment consisted of a restricted fluid intake of about 700 cc. daily and sometimes even as low as 350 to 400 cc., a diet low in salt for the purpose of increasing diuresis, a reduction diet for patients who were overweight, together with exercise, massage and light therapy to stimulate vasomotor tone. Of 157 patients who remained under this regime in the hospital for a minimum period of two weeks,

Dederding reports that the vertigo and nystagmus disappeared in all but 7 and that the hearing improved in all but 26. Further details as to the permanency of this relief of symptoms after the patients left the hospital are not available, and knowing that there are remissions of variable periods in this symptom complex as the result of various forms of treatment and also from no known cause we do not know how long these apparent "cures" persisted after these patients returned to their homes.

Furstenberg, Lashmet and Lathrop,[3] believing to be incorrect the deductions of Mygind and Dederding that water retention was the cause of the Ménière Syndrome and that when the body accumulates water sodium is also stored, directed their treatment to a reduction of the body sodium. By a series of experiments they proved that (1) the retention of water and sodium would produce an attack, (2) the retention of water without sodium would not produce an attack, (3) the loss of water with a retention of sodium produced an attack, and (4) the loss of water and sodium did not produce an attack. Furstenberg, therefore, put his patients on a salt-free diet to limit the intake of sodium, and on ammonium chloride, an acid producing salt, to encourage an increased excretion of sodium while allowing the patients all the water desired, and he reported very favorable results. He concludes that the water intake need not be considered if the concentration of the sodium ion in the body is kept at a low level.

Favorable results with the Furstenberg regime were also reported by Brown[20] and by Bartels.[21]

Walsh and Adson[6] report upon a series of 186 cases, of which total 152 were treated with a low salt diet with or without the addition of medication for salt elimination. Because of complaints that ammonium chloride disturbed the appetite and digestion of their patients they began to employ potassium nitrate for the same purpose. The work of Keith,[22] Talbot[22] and others showed that this salt also causes an elimination of sodium from the body. Walsh and Adson found potassium nitrate as efficacious in the treatment as ammonium chloride.

Considering the 152 patients treated with a low salt diet with or without the addition of sodium-eliminating medication they found that 52, or 34 per cent, experienced complete relief of the vertigo, 43, or 28 per cent, experienced variable degrees of improvement, and 57, or 38 per cent, experienced no relief whatever.

With regard to the deafness Walsh and Adson[6] note that there was improvement in 68 per cent of the cases with unilateral deafness and in 53 per cent of the cases with bilateral involvement.

In regard to the patients who did not show any improvement under this regime they are not entirely sure that the patients carefully followed the treatment when they returned to their homes. Furstenberg also draws attention to the difficulty experienced in keeping patients on a salt-free diet after they leave the hospital. A number of the patients of Walsh and Adson who had experienced complete relief from their symptoms while under treatment stated that their symptoms returned when they added salt to their diet.

Morsch[29] presents the case histories of four patients with the typical symptoms of the Ménière Syndrome who also presented symptoms of avitaminosis for Vitamins A and C. He reports an improvement in the vertigo, tinnitus and deafness of all four after intensive vitamin treatment.

In January 1940 Shelden and Horton[19] presented a preliminary report on the treatment of the Ménière Syndrome with histamine. They believe that the "site of the lesion undoubtedly is in the inner ear itself, and that the disturbance of function of specific regions alone or in combination determines the variations in the syndrome". They also believe that the factor most likely responsible for the symptoms is a local alteration in the permeability of the capillary wall with secondary local edema. Histamine is an important agent affecting capillary permeability. The action of histamine in controlling the symptoms of the Ménière Syndrome was accidentally discovered by one of them (Horton) while treating vascular headaches with this drug. A patient with vascular headache and who also had the symptoms of the Ménière Syndrome was relieved of all symptoms under histamine treatment. Of the 15 patients included in their report 4 were treated by the subcutaneous route and 11 intravenously. With the exception of 3, all of these patients had been on the Furstenberg regime with either ammonium chloride or potassium nitrate medication for several months but without benefit. They report spectacular results in all 15 patients, and no ill results were noted from the histamine medication. The details of the treatment can be found in their publication.

In a personal communication dated May 9, 1940, Shelden[23] states that they have continued to use the intravenous histamine medication with gratifying results and with no other modification

than an occasional administration of small amounts of histamine subcutaneously (.1 to .2 mg. two or three times weekly) to prevent recurrence.

In commenting on the paper of Shelden and Horton, H. H. Woltman[24] states that because of its value and ready applicability he does not believe that the Furstenberg treatment will be supplanted by the histamine treatment, but that this latter medication is of great value when the vertigo and vomiting are extreme, calling attention, as others have done, to the fact that the Furstenberg regime does not operate immediately.

The surgical treatment of the Ménière Syndrome aims at the interruption of impulses arising within the labyrinth and passing to the brain. It is predicated on the assumption that the lesion causing the symptoms is located either in the labyrinth or in the acoustic nerve. This surgical intervention consists in either severing the eighth nerve or its vestibular portion intracranially or in an obliteration of the end organs in the labyrinth.

According to Dandy the operation for section of the eighth nerve was designed by Krause of Berlin for lesions of cranial nerves. It was used by him for this purpose and by Frazier[25] in 1907 and Ballance[25] in 1908 for sectioning the eighth nerve because of tinnitus but without success. It is Dandy's claim to have used this operation for the first time in Ménière's Syndrome in 1927. His modification of Krause's operation is the preliminary drainage of the cisterna magna to produce more room in the operative field and to obviate damage to the cerebellum. Since 1927 he has operated upon more than 250 cases without a fatality. If the hearing is very bad he sections the whole nerve, if relatively good, only its vestibular portion.

McKenzie and McGregor[11] have shown that while the separation of the cochlear and vestibular fibers is fairly complete after the acoustic nerve leaves the internal auditory meatus, there is some intermingling of these fibers along the line of separation. Dandy, however, points out that quite a large number of cochlear fibers can be severed during the differential section of the vestibular nerve without further damage of the hearing. If the higher tones have not already been lost due to the disease it is the high tones which are damaged by a partial section of the cochlear nerve, and this fact suggests to Crowe[5] that localized areas of Corti's organ and only certain fibers in the cochlear nerve transmit high tones, while low tones are

not specifically localized in the cochlea and are transmitted by any undamaged fibers of the cochlear nerve.

Section of the entire eighth nerve or its vestibular portion usually eliminates the violent attacks of vertigo, as pointed out by Dandy,[4] McKenzie,[11] Walsh and Adson[6] and others. That it does not always do so is indicated by the figures of Walsh and Adson who state that of 20 patients who had had a section of either the whole nerve or its vestibular portion 15 were able to resume their work and of the remaining 5, 3 were highly nervous women. They suggest that in those cases which do not gain relief from the total nerve section the condition is bilateral and the opposite vestibular nerve should be sectioned, but in most of the cases the vertigo, if not completely eliminated, is improved to the extent that the individuals can resume their occupations.

While section of the eighth nerve or its vestibular component offers great possibilities of relief for the intractable and disabling vertigo, the same cannot be said for the tinnitus, which is admitted by all neuro-surgeons doing this type of operation. This fact alone would argue against the exclusive localization of the lesion in the labyrinth.

Dandy presents the following postoperative sequellae:

1. He has not lost a case by death. McKenzie,[11] however, reports one postoperative death from wound infection and Hallpike[17] reports on the microscopic examination of the temporal bones of two individuals who died following an intracranial nerve section.

2. In two of his earlier cases the seventh nerve was severed.

3. In three patients a late facial paralysis developed which cleared up spontaneously.

4. Transient postoperative diplopia occurred in four cases and he thinks this is of the same nature as the transient diplopia occasionally seen during the Ménière attacks.

5. Transient nystagmus occurred rather frequently after operation.

6. Transient vertigo after section of the nerve occurred in the majority of his cases. This was mild in most of the cases but severe in some. It usually began on the first postoperative day, reached its peak in 48 hours and was gone in from seven to ten days. After this it might recur, on turning the head, for a year or more.

Crowe[5] investigated the hearing of 72 patients on whom Dandy had performed a differential section of the vestibular nerve and found that following the operation:

1. The deafness in the affected ear was worse in 22.
2. The deafness in the affected ear was unchanged in 36.
3. The deafness in the affected ear was better in 14.

The average elapsed time between the operation and the last examination was 2.2 years.

For several years, otologists, following the same line of reasoning as the exponents of nerve section, namely, the interruption of abnormal impulses from the end organ, have been practicing destruction of the sensory epithelium of the end organ. Mollison[26] accomplishes this by exposing the prominence of the external semicircular canal by a simple mastoid operation and injecting a few drops of absolute alcohol into the membranous canal after removal of its bony covering. He reports that 74 per cent of patients so operated upon expressed themselves as cured, 10 per cent as not benefited and 16 per cent could not be traced. There were no fatalities from this procedure, but one case developed a transient facial paralysis which cleared up in three weeks. Berggren[27] uses the same method of alcohol injection though through a different approach. He makes a flap of the entire posterior one-half of the membrana tympani, turns it upward and forward, and after scarifying the mucous membrane over the promontory he perforates the bone in this region with a dental burr.

Putnam[28] suggests drilling into the superior canal with a burr through a subtemporal approach and destroying the labyrinth by coagulation.

Physiologically, there can be no essential difference between destroying the labyrinth in this fashion and cutting the nerve fibers arising within it, providing destruction is complete in both cases. But the destruction of the labyrinth by chemical or thermal means must be reserved for those cases in which the hearing is gone or nearly so.

SUMMARY

In the Ménière Syndrome we have a fairly common condition affecting middle life, characterized by a triad of symptoms, vertigo, tinnitus and deafness, of unknown etiology and little known path-

ology. In the etiology more serious consideration must be given to allergy, avitaminosis and disturbances in the endocrine balance than has been accorded to them hitherto.

If the pathological condition of the temporal bone as shown by Hallpike is correct for every case, then the main pathological feature of the condition is a waterlogging or edema, as is maintained by Mygind,[2] Dederding,[7] Furstenberg[9] and others. This condition is probably a metabolic disturbance but whether a disturbance in the water balance as Mygind and Dederding[2] maintain or a disturbance in the metabolism of the sodium ion according to Furstenberg[9] or both is not entirely clear.

The operation of this disturbance in metabolism seems to find its greatest expression in the labyrinth where it initiates the attacks of vertigo, the deafness and probably also some of the tinnitus. That not all of the tinnitus originates in the end organ is evidenced by the fact that it persists in many cases after total destruction of the labyrinth or a severance of its nerves. May it not be that this same waterlogging process is operating both in the end organ and also in the central cochlear nuclei?

The success of the dehydration treatment of Mygind and Dederding[2] and the sodium elimination treatment of Furstenberg[9] as well as the more recent treatment with histamine when carried out under proper hospital supervision suggests that the patient should be given the opportunity of trying medical treatment before submitting to surgical intervention.

Surgical intervention should be reserved for those patients who have not responded to a medical regime, to those patients who for economic or other reasons cannot be kept on a medical regime, and for those patients whose occupations are such as to preclude the possibility of any return of the vertigo because of carelessness in following a medical regime. Of the surgical measures proposed it would seem that total section of the acoustic nerve is indicated for those whose hearing in the affected ear has fallen below a usable level and that the differential section of the vestibular portion of the nerve is the operation of choice for those with usable hearing. These operations should not prove hazardous in the hands of the competent neuro-surgeon.

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VIII

WAR DEAFNESS AND ITS PREVENTION*

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BALTIMORE

All impaired hearing that develops in time of war might logically be included in a definition of War Deafness. The term is usually understood, however, to mean more particularly those injuries to hearing that are caused by acoustic trauma. For the most part, the present paper is about War Deafness caused by the two principal forms of acoustic trauma encountered in modern warfare, namely, recurrent extremely loud noise for long periods of time and detonation waves from nearby explosions, and about practical measures for the prevention of such injuries.

No cause of impaired hearing is peculiar to war times. During war, however, the incidence of injured hearing from several causes is markedly greater in military personnel than in the corresponding age-group of civilians in time of peace. Prominent among the causes with an increased incidence are: epidemic spinal meningitis complicated by labyrinthitis, the several forms of otitis media and their complications, direct wounds of the head with missiles, rapid changes in altitude in combat flying and in training for the same, and the two forms of acoustic trauma already mentioned.

Before proceeding to the consideration of acoustic trauma, the other causes mentioned will be briefly discussed, especially from the viewpoint of prevention of impaired hearing.

Labyrinthitis as a complication of epidemic or meningococcic spinal meningitis seems to occur less frequently than it did previous to the advent of treatment with sulfanilamide and related compounds. Although statistical data are not available with respect to deafness, chemotherapy seems to have reduced markedly not only the mortality rate of this disease, which is dreaded in all armies, but also markedly to have reduced the incidence of serious complications in those who survive an attack in civilian life. When statistical data

*From the Otological Research Laboratory, The Johns Hopkins Medical School. Read at the meeting of the Middle Section of the American Laryngological, Rhinological and Otological Society, at Chicago, January 27, 1941.

for the present conflict become available it should be possible, by comparison with similar data for the first World War, to evaluate the importance of this preventive of War Deafness.

Otitis media and the complications of its several forms certainly must be rated an important cause of impaired hearing in military personnel, and all measures that reduce the incidence of otitis media, shorten the duration of attacks or lessen the severity of complications are at the same time measures that reduce the incidence of handicapping degrees of War Deafness. In troops, as in civilians, otitis media is most commonly a complication of an infection of the upper air passages. All measures that reduce the incidence or the severity of the attack of the common cold, of sinusitis, of pharyngitis, of influenza, of grippe, of measles, scarlet fever, diphtheria, etc., at the same time reduce the incidence of War Deafness caused by otitis media and its complications. Many of the men who eventually have to be discharged from military service because of otitis media and its complications should never have been accepted in the first place. Better physical examinations of candidates is the logical method to prevent numerous instances of War Deafness and also to prevent the economic loss incurred in their wasted training, their hospital care, and eventually their disability pensions.

The use of steel helmets instead of the cloth hats and caps of earlier wars materially reduces the number of direct wounds of the head that would otherwise occur, by shell fragments, rocks and other secondary missiles, as well as by bullets themselves. Without doubt this prevention of direct wounds of the head also serves to prevent the impaired hearing that would result from certain of the wounds, whether by direct penetration of some part of the ear or indirectly from a basal skull fracture including the temporal bone. Every improvement in metal helmets and in other means of protecting the head from missiles may therefore also be regarded as a method of prevention of impaired hearing. Even such a simple improvement as turning down the sides of the brim of the helmet, without increasing its weight, has probably already prevented many cases of injured hearing.

Rapid changes in atmospheric pressure constitute the principal threat to the ear in flying. In modern military planes not only is the cabin enclosed but the ears of the personnel are covered by the receivers and close-fitting rubber ear-caps of radio or of intercommunication apparatus; therefore even under combat conditions the danger of acoustic trauma is completely secondary to that of acute tubal occlusion. To prevent serious injury to the hearing in flying

it is necessary to maintain the patency of the eustachian tubes. It is very difficult to do this, even for a man with an entirely normal nasopharynx, when the altitude is changing at rates of from one to five or more miles per minute, and especially so when he is extremely busy with several other things. A satisfactory solution to the problem of the prevention of this important type of War Deafness has not been found.

ACOUSTIC TRAUMA*

Artillery and navy men have for centuries been subjected to acoustic insults to the ear, but previous to the first World War infantry troops seldom were exposed to sounds louder than the explosions of shrapnel shells at a considerable distance from them. Trench warfare brought about the intensive use of high-explosive shells and of hand grenades, with the result that infantry as well as artillery and navy personnel were frequently exposed to injuriously loud sounds and detonations. In the Spanish Civil War large numbers of civilians also were exposed to acoustic trauma in the shelling and bombing of cities; Collier reports that Trueta saw in Barcelona 175 civilians with detonation injuries to the ear. In the present conflicts, both in Europe and in China, the danger of acoustic trauma must be considered in planning for large portions of the civilian population as well as for the entire military personnel.

Acoustic trauma causes more impairment of hearing for high tones than for low tones. In the milder cases, after recovery from the fatigue element, the impairment is limited entirely to high frequencies, above 2048 or 2896 cycles per second, and the hearing for conversational voice is good except for the highest of the sibilant sounds. A high pitched buzzing tinnitus is frequently complained of. This clinical picture is well known in civilian life as the early stage of an occupational deafness, and also is frequently found in men who do much hunting or who have accidentally had an ear close to the muzzle of another's gun when it was fired. Fortunately, this milder type of damaged hearing is the one most frequently caused by the acoustic trauma of war. The lesion probably is in the basal turn of the cochlea, and consists of a more or less extensive destruc-

*Very little new or important has been added to the knowledge of acoustic trauma or its prevention since the period of the first World War. The basis for many of the statements made in the present brief review can be found, by those interested, in my papers in 1917, 1918 and 1919. In these papers the topic is presented more fully than here, and numerous references to literature are given.

tion of the organ of Corti and atrophy of the nerve fibers. There is no effective treatment for this lesion.

A single detonation can cause permanent severe impairment of hearing for all tones, with loss of usable hearing for even the loudly spoken voice. Many cases were reported in the first World War in which the combination of circumstances was such that the deafened individual was not hit by any shell fragments or even by secondary missiles, although frequently he was rendered temporarily unconscious by the powerful detonation wave. The tympanic membrane is often torn by the very intense detonation waves, in which the air pressure in the external auditory canal may be compressed, for a very brief period of time, to several times the normal atmospheric. Unless infection supervenes, the torn tympanic membrane usually heals promptly. Otitis media in these cases frequently is caused by injudicious attempts to clean the ear before the tympanic membrane has had a chance to heal. The extent of the organic lesion of the inner ear in cases of severe detonation trauma has not been established by histologic examination of human temporal bones, so far as I am aware. From animal experiments made by several investigators it seems probable that in man also there is extensive damage to the cochlear end-organ and extensive atrophy of the nerve. Some patients have had severe vertigo, nausea and even vomiting for days or weeks after the accident, but it is not safe from this functional disturbance to conclude that there is an organic lesion of the vestibular end-organs.

The impression was gained from clinical observation before and during the first World War that rupture of the tympanic membrane tends to protect the inner ear. A similar impression was gained from the animal experiments which I made in studying the effectiveness of devices to protect the ear from acoustic trauma; the cochlear lesions were in general more severe and extensive in the animals whose tympanic membranes remained intact.

To prevent damage to the ear by acoustic trauma the intensity of the waves, whether sound or detonation, has only to be reduced sufficiently; the waves do not have to be stopped entirely. Many methods of protection of the ear have been proposed and many devices have been made for this purpose. Most of them are unsatisfactory, for one reason or another.

A finger tip placed firmly over the end of the external auditory canal sufficiently reduces the intensity of the air waves that reach the tympanic membrane, and this simplest method of all would be quite

satisfactory if one could always anticipate the need and had no other use for either hand. It is a method frequently resorted to by artillery men at target practice.

Solid obturators of all types, if they fit the external canal properly, sufficiently reduce the intensity of sound and of detonation waves, but long continued wearing of them causes most people marked discomfort, their use favors the development of infections of the external canal, etc. In addition, solid obturators are entirely unsuitable for use by those whose duties require that they be able to hear ordinary sounds well at all times. The latter objection applies also to the use of cotton or of "waste" soaked with oil or with vaseline, both of which effectively protect an ear from acoustic trauma.

With dry cotton tightly packed in the external canal ordinary sounds, such as commands, warning cries, etc., can be fairly well heard, but dry cotton is very ineffective in stopping even moderately intense detonation waves. This fact has been repeatedly stated in the literature on the subject, but nevertheless dry cotton is still the most commonly used method of trying to protect the hearing. In my experimental testing of the efficiency of various devices, histologic examination proved that the ears of animals protected by dry cotton were severely injured, and the physical method used to rank the devices in order of relative efficiency showed dry cotton to be the least effective of all in stopping small detonation waves.

Water-soaked cotton soon dries out enough to have air-containting spaces and loses whatever effectiveness it may at first have in protecting the ear.

Obturators made of wax mixtures that can be shaped at body temperature to fit the auditory canal closely are effective in stopping injurious intensities of detonations and are more comfortable to wear than are solid obturators, but with them ordinary sounds cannot be heard sufficiently well for most military duties.

Several devices designed to permit good hearing of ordinary sounds and at the same time to stop detonation waves have been made. Some have been patented and produced commercially. In general, these devices utilize one or the other of three principles: 1) the elastic properties of rubber, 2) a thin diaphragm backed up by perforated metal discs, with the assembly mounted in a hard rubber or vulcanite tube shaped to fit into the external auditory canal, 3) a quick acting valve mechanism mounted like the preceding.

All devices based on either of the latter two principles have, in common with solid obturators, an objection for military use that has not yet been mentioned. If struck by a projectile which might otherwise inflict only a minor superficial wound, the device is shattered and the fragments may cause serious wounds. Some of the devices perform rather well under laboratory conditions, but under field conditions it would be impossible to keep them clean enough to be of any value.

In 1917 and 1918 I made laboratory tests of the efficiency of many devices and methods that had been proposed for protecting the ear from detonation trauma. Some of the devices tested were not included in the published reports. It is unnecessary at this time to describe the techniques used, except to say that the detonation wave used in the animal experiments regularly ruptured the tympanic membrane of an unprotected ear and caused severe damage to the organ of Corti. To create the detonation wave a ball cartridge was fired from a .45 caliber pistol held with the muzzle six inches in front of the ear and pointed so that the projectile passed alongside the animal's body.

The device which proved best, all things considered, in my laboratory tests also was rated as highly efficient in the tests made by Trible and Watkins, of the U. S. Navy Medical Corps, near the muzzles of large caliber naval guns. The war ended before the device had the field trial recommended by the Surgeon General of the U. S. Army, so that its actual performance under combat conditions was not established, nor, so far as I am aware, has it ever had a field trial in any army or navy. Because no device has since been made that gives better promise of practical usefulness, this one merits description and consideration again at the present time.

The device was manufactured by a British firm which went out of business shortly after the end of the first World War. It had the rather elaborate name of The Scientific Ear Drum Protector "Tommy", and was advertised in the public press in England. The "Tommy" consisted of a hollow soft rubber spherical bulb with an opening on one side, and the short necked opening was surrounded by a soft rubber flange. The external diameter of the spherical part was about 9.5 mm. and of the flange, about 11.5 mm. The wall was about 1.5 mm. thick.* Proper insertion of the "Tommy" into the end of the external auditory canal was easily accomplished; the

*Both in shape and in thickness of wall the "Tommy" differs materially from the rubber ear-stopples commonly used by swimmers, which give but little protection from detonation trauma.

flange kept it from going too deeply, and one size fitted almost all auditory canals of adults without causing an uncomfortable pressure. They could be cleaned easily wherever soap and water were available, and the risk of fragmenting to form secondary projectiles was almost nil.

The cost of the "Tommy" was only one shilling a pair, retail. In quantity they could doubtless be produced for only a few cents a pair.

Voice sounds, even when quite soft, could be easily heard when wearing "Tommies"; they took the "sting" out of intense sounds and effectively stopped detonation waves.

In conclusion, I wish to point out that to prevent War Deafness it is necessary not only to have the required technical information but also to have suitable action taken by those in authority. This is true both for military personnel and for civilian populations, and it applies to each of the causes discussed in this review and to those causes not mentioned.

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IX

OTOLOGICAL RESEARCH AND ITS RECENT CONTRIBUTIONS TO CLINICAL OTOLOGY*

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Although it is entirely improper to require any research effort to proceed on a set schedule of actual accomplishment, nevertheless from time to time pause must be taken for appraisal of present achievements. The purpose of this discussion, therefore, may appropriately be considered as a critical summary of a ten years' period in otological research. This time interval is not set arbitrarily. Before 1930 such laboratories as were in existence confined their efforts to studies of clinical pathologic otology and the more obvious aspects of auditory function such as reasonably reliable methods of measuring hearing loss. Before this time no adequate means for research in the physiology of hearing or its pathologic physiology were available. Twenty years ago an analogous situation existed in the general field of anatomy, when, largely through the effort of the Hopkins group, led by Weed, gross anatomy ceased to be a matter of importance and gave way to intensive effort in physiological anatomy. Other fields of medicine involving more vital structures than the ear offered problems for physiological research which were carried out by many scientific groups, and, as a result, today our diagnostic and therapeutic armamentaria in many fields of medicine are immeasurably broadened. In other words without a specific knowledge of normal function, familiarity with normal and pathologic structure is relatively unimportant.

By its very nature and position in the body, the ear has baffled any rational study of its specific activities until recent years. Since it serves to transmit to the brain acoustic stimuli, analysis of audi-

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tory function has always been based on laws of physical acoustics. However valid such hypothecation may have been or still may be, it cannot carry with it the conviction inherent in properly controlled physiological experiments.

This particular point warrants further elaboration. For many years American otologists have been stultified, so far as individual effort and expression of purpose is concerned, by an extraordinary subservience to European reputation and dicta. There can be no question that during a certain period there came from Europe the only advances in knowledge in the field of otology. Helmholtz, Pulitzer, Barany and many others forced Americans, whether spurred by a desire for true scientific knowledge or simply to gain local prestige, into the general hegira from American otologic incompetence. This fetish maintained itself in an utterly unreasoning manner, and no longer ago than ten years, American otologists who had received some of their training in European clinics were still sending their patients abroad for consultation and treatment. Under such circumstances there could be no possible thought of progress in a group so completely under the dominance of foreign rule and guidance. Strangely enough this scientific background insinuated itself into the field of therapy also, and returning Americans brought back with them not only microscopic slides, prepared purely for commercial purposes, but routines of treatment, many of which still form the background of much otologic therapy. There was apparently no escape from this all powerful influence.

Crowe in Baltimore, though imbued with the background of a European training, was the first in America to divorce himself from this dependence on foreign microscopic material. Today the Hopkins collection of serially sectioned temporal bones of patients with recorded otologic and audiometric data is unequalled in the world, both from the standpoint of perfected histologic technique and that of wealth of pathologic material. This high achievement is due largely to the unflagging efforts of the director of the Hopkins Laboratory, Stacy Guild.

Though not in direct chronologic sequence, an important change in the field of otologic therapy has taken place in the past four years. There is probably no person in the United States with a hearing loss of any consequence who has not been subjected to the very trying and often painful procedure of catheterization, bougienage, and inflation of the eustachian tubes either by a private physician or in a hospital clinic. This is a direct and specific contribution from the European school of otology. Indeed in the late 1920's in

various European centers hospitals, private and general, were flourishing where eustachian bougies were inserted in the morning and left in place until a time when the attending physician could conveniently withdraw them in the late afternoon. The entire procedure was carried out with no possible regard either to the patient's comfort or to any reasonable consideration of asepsis. Happily one otologist spoke with complete authority and conviction, decrying the promiscuous application of this procedure to deafness. And yet it has and is still being used without regard to cause, age or possible benefit and attendant hazard. Friesner,[1] in 1937, showed that under routine circumstances the procedure of bougienage and inflation had never been and anatomically could not be a matter of any possible benefit in an established adult deafness. This exposition was revolutionary but, unhappily, has as yet failed to wipe out the futile practice.

The oft-repeated slogan, "If it will not improve, at least the impairment will not progress", is one of our otologic black marks. Bunch[2] has shown that in the great majority of deafened ears, hearing impairment is not progressive beyond a certain level. Cases observed in this laboratory have never shown a rapid decline in hearing provided the impairment had reached a relatively critical point. This statement is made with all due consideration to the facts involved. It is, or should be, well known that at certain levels of impairment the individual clings to the feeling that his hearing is relatively normal. At this point a further loss of 10, 15 or 20 decibels in any part of the speech frequency range renders him definitely hard of hearing. He will say the hearing loss occurred over night, during a cold, following extreme fatigue, or after exposure to acoustic trauma of exaggerated force. Actually some impairment has been present probably for years. The extra 10 decibels was the measure between possible auditory competence and isolation. But beyond this point of personal realization of a handicap the vast majority of individuals seldom pass unless exposed to the superimposed factors of acute infection, trauma, misdirected therapy, or old age (Chart I).

INHERITED DEAFNESS

When exposed to questioning, the average patient will volunteer information in regard to a familial tendency. Parents, aunts, uncles, and members of earlier generations have exhibited the same unhappy defects. Analysis fails to disclose anything more important that that one parent became deaf in his or her old age, an aunt or

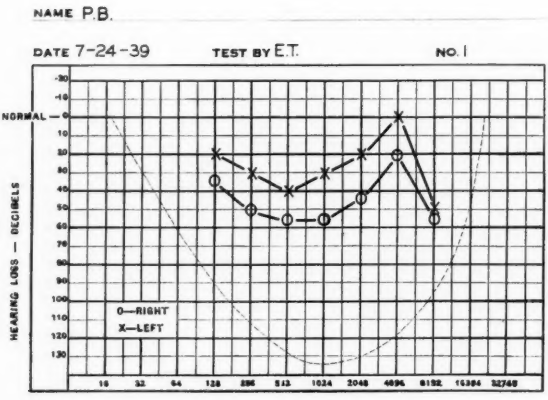

Chart I. This audiogram illustrates the effect of a superimposed factor upon an established hearing loss. The patient was unconscious of any hearing impairment until the right ear was exposed to a loud telephone click, following which the ear pained severely and deafness was apparent. However, there was obviously a loss prior to this accident as evidenced by the level of the opposite ear. In addition both tympanic membranes were retracted and thickened and showed every evidence of a chronic catarrhal otitis media. The additional loss of ten decibels in the speech frequency range made the difference to the patient betwen normal and impaired hearing.

uncle was deaf from childhood, a brother or sister had discharging ears for years. A great many family histories probably have little to do with the presenting case of deafness. Such a family tree is the following (Chart II).

Schools for the deaf have student bodies largely made up of supposedly congenitally deaf children. This is an entirely erroneous supposition. Only a small percentage of these children present true congenital deafness. The criteria for this particular disability are specific. The mother and father must both be deaf from birth; the child may or may not be so, but if the deafness is truly congenital the heritage must be clear. A survey (unpublished) of the families of 487 children at the Pennsylvania School for the Deaf revealed the fact that only 13 children were born of such deaf parents; the rest of those classified as congenital were born deaf or acquired deafness in the neo-natal or early infantile period for no explainable reason. The conclusion drawn from these data was that some biologic defect,

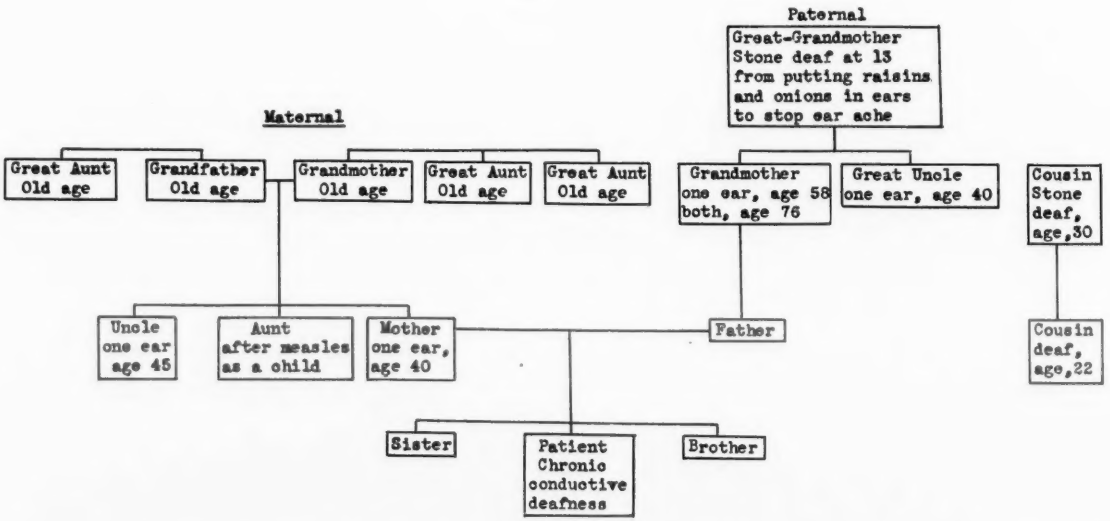

Chart II. A family tree recording deafness in three generations. Of 17 individuals 14 had impairment of hearing. The patient, a young woman 30 years of age, naturally felt that her own impairment was of necessity hereditary in character. However, careful examination of ascribed cause and character of the deafness in this individual's case fails to demonstrate any possible familial factor other than frequent old age deafness, except that appearing in second cousins once removed. The stigma of hereditary deafness has a dangerous psychological effect on many patients and as in this instance is often unwarranted.

present at birth or manifesting itself shortly thereafter (seldom after three years), was responsible for the so-called congenital deafness.

For many years early deafness has been associated with the incidence of the various contagious diseases, scarlet fever, with or without acute middle ear involvement, diphtheria with its possible late toxic effect upon the auditory nerve, and mumps, responsible for no really explained reason. A recent analysis[3] of the etiology of deafness in a deaf school population lists 17% as being caused by epidemic meningococcus meningitis. The immediate reaction is one of incredulity. An explanation, however, is readily apparent. With modern therapeutic measures, anti-sera and sulfanilamide drug treatment, lives are saved but at the expense of special sense organs, in this particular instance most frequently the ear.[4]

Disease of the nasopharynx has quite lately been revived as a contributing cause to youthful and adult deafness. There can be no question that overgrowth or infection of adenoid tissue in early life is responsible for a certain percentage of hearing impairment in the pre-school and school child. Early correction of the difficulty will often produce dramatic results. However with each passing year the benefit of treatment becomes more problematical and less effective.

It is a matter of note that the present great interest in the rational therapy of deafness has followed rather than preceded the revival of experimental investigation of the sense of hearing. The reason for this state of affairs is obvious. Ten years ago direct methods for studying auditory function were lacking so far as actual laboratory experimentation is concerned. Microscopic study of human pathologic material was the basis for all therapy. Function was assumed rather than proven.

Probably the outstanding achievement of this microscopic pathology era was the report of Crowe, Guild and Polvogt[5] upon the etiology of high tone loss. These authors, with a wealth of material available nowhere else in the world, showed that with varying degrees of atrophy of the organ of Corti, nerve, stria vascularis, and sulcus cells in the basilar turn of the cochlea, high frequencies of hearing were invariably lost. As the atrophy proceeded towards the mid-modiolar region, more of the higher frequencies were lost. A spatial or place theory was postulated. The fact that the great concentration of spiral ganglion cells lay in this area was not considered. No satisfactory explanation for so-called "tone island loss" was presented nor in any single instance was there demonstrated low fre-

quency loss as a result of apical atrophy. The relationship between numerical loss of ganglion cell and frequency perception is a matter which still permits of speculation.

Into this strictly morphologic field of investigation was injected in 1931 the dramatic experimental method, first described in detail by Wever and Bray[6] and subsequently known in the literature as the Wever and Bray phenomenon of the cochlear response. Although the response had been observed before by Adrian[7] its significance from the standpoint of the normal and pathologic physiology of hearing was certainly not appreciated.

Immediately after the announcement of these observations, based upon the measurement of changes in electrical potential in the cochlea and auditory nerve resulting from acoustic stimulation of the ear of experimental animals, various investigators established laboratories for the study of the physiology of hearing. Interest was centered at first upon the establishment of the fact that the electrical changes were truly a function of the auditory apparatus and not simply an electrical artifact. Agreement was finally reached by even the most sceptical that the response was definitely initiated by the perceptive mechanism of the ear, that it was dependent upon the viability of the total organism and that it could definitely be used as a measure of functional ability and integrity of the end organ.

The experimental approach in the various laboratories was markedly diversified. In some, abstruse problems dealing with the nature of auditory response as measured by electrical changes in the cochlea and auditory nerve were investigated. It was shown by some observers, for instance, that experimental atrophy of the nerve and spiral ganglion cells could be produced with no impairment whatever of the cochlear response from the round window or from the cochlear wall, providing the organ of Corti was intact. Theoretical consideration dominated the early studies. Localized damage of the organ of Corti by the use of a high frequency cautery applied to holes drilled partially through the bony wall of the cochlea was first employed to demonstrate experimentally the clinical phenomenon of "tone island loss". Specific areas of the cochlear spiral were damaged, but the electrical responses resulting did not support the original thesis.[8] Hallpike,[9] appropriating this technique, showed that high frequencies were most intense at the base and that the reverse was true at the apex of the cochlea.

About this time the conditioned reflex technique was revived, and Culler,[10] reporting in 1935, mapped out an extraordinarily con-

vincing tone localization for the complete cochlear spiral. Other investigators[11, 12] of this conditioned reflex collaborated with laboratories studying electrical potential changes, and it was found that fundamental responses in experimental animals were the same under the two conditions. Controversy as to the actual nature of the electrical response proved of no real importance. Bast[13] maintained that polarization across Reissner's membrane was the essential factor. Howe[14] reported studies on congenitally deaf albino cats with normal Reissner's membrane and an undeveloped organ of Corti. In these animals there was no electrical cochlear activity. Indeed the fact was demonstrated time and again that the one essential for demonstration of the effect was a normal organ of Corti, regardless of any other structure in the perceptive mechanism. Later, Lurie[15] from the Harvard Laboratory postulated auditory response in relation to the integrity of individual inner and outer hair cells. He even claimed to be able to hypothecate an audiogram on the basis of histologic study of serial sections of the cochleae of guinea pigs.

A completely novel and ingenious approach to the whole problem of the physiology of hearing was developed and elaborated by Kobrak.[16] Using guinea pigs, he found that contraction of the tensor tympani muscle as a result of acoustic stimulation might serve as a measure of auditory response. This quite obviously was a limited method due to its direct mechanical implication. Previously Hallpike[17] had called attention to the fact that activity of this muscle was eliminated by anesthesia. However even under profound ether anesthesia the muscle could be observed to contract following tactile stimulation of the external auditory canal. Similar experiments carried out upon decerebrate animals revealed an identical effect. The tensor muscle and probably the stapedius too function only when the acoustic stimulus approaches an intensity which is essentially a tactile one. All experimental evidence points towards the protective function of these two intrinsic muscles of the ear. If intact they tense the ossicular chain and thus dampen the effect of acoustic trauma. When the ossicular chain is deprived of protection by division or paralysis of these muscles, acoustic trauma exerts its unimpeded force upon the delicate organ of Corti. The observed microscopic damage is a complete shattering of this important sensory receptive mechanism.[18]

No more complete resumé of purely experimental findings could possibly be compiled than that of Wever[19] in his paper, "The Electrical Responses of the Ear". However, the title of this present report implies a specific application of experimental findings to the

problems of clinical hearing loss. Admitting that certain criteria of sound conduction must, at the moment, be accepted as final, other facts of clinical significance cannot well be gainsaid. The ultimate value of animal experimentation here as in every other field of medicine must be its careful and thoughtful application to the particular clinical problem involved.

In 1931 there was established at the Johns Hopkins Medical School a laboratory for otological research using the Wever and Bray technique. The purpose of the investigation was primarily to establish the normal physiology of hearing, to correlate with it pathologic physiology resulting from experimental procedures and to develop, if possible, methods whereby an established hearing loss, either in animals or human beings, might in some way be alleviated or improved. Various facts to be elaborated upon were definitely established. It is interesting to note that they are still valid ten years after the original exposition. The function of the round window was first explored[20] as no satisfactory explanation of its function was at that time accepted. Acute and chronic animal experiments showed that this cochlear window with its membrane was, under any ordinary acoustic circumstance, relatively a passive part of the total mechanism. However, under the stress of extreme acoustic stimulation or even ordinary stimulation with the round window blocked, auditory response was greatly enhanced. Of particular significance was the improvement in acuity of normal ears following fixation of the round window in experimental animals. Culler[21] and others failed to confirm these findings due to lack of understanding of the particular technique involved.

At this point digression may be permitted to elucidate various incompatible reports relative to fixation of the round window membrane. Culler used chewing gum wrapped in gauze. So delicate is the round window membrane that any chemical substance other than normal saline solution will exert an immediate and deleterious effect upon that part of the organ of Corti in the basal coil. In fact, cold, heat, hypertonic or hypotonic saline solution, any or all drugs in mildly concentrated solution, affect immediately the adjacent organ of Corti through this delicate membrane. Again, however, this situation probably seldom develops in the life history of the hearing mechanism.

Clinical application of these experimental findings has been appraised in 38 instances, and as reported in a four-year period of observation,[22] appreciable improvement in the hearing of deafened

individuals has been obtained. Since the publication of these reports, further observations have been made relative to blocking the round window in two patients with chronic suppurative otitis media. In both of these patients the tympanic membrane was completely absent, and the middle ear cavity was filled with granulation tissue except over the round window niche. Cotton moistened with normal saline solution packed into the niche caused a marked improvement in hearing. The case history and audiometric studies of one of these patients follows:

REPORT OF A CASE

The patient was first seen September 23, 1940, complaining of deafness and discharge from both ears. She is twenty-eight years of age and a trained nurse by occupation. Her family history is negative. The past history and present illness are concomitant. Following an attack of measles at the age of four years, bilateral suppurative otitis media developed and has been present almost continuously since that time with occasional remissions. Tonsils and adenoids were removed at the age of five years. A radical mastoid, left, was performed in 1925 and an operation for recurrent tonsillar tissue in 1932. For the past three years she has had symptoms of vertigo relative to an involvement of the left labyrinth. There is no neurologic evidence of any intracranial disease. There is a mild bilateral tinnitus.

Hearing in the left ear is practically absent and markedly reduced in the right. The patient wore a bone-conduction hearing aid with reasonable success. At the present time the strain of carrying out her professional duties with the hearing handicap is having a marked effect upon her general health and psychological reactions. Her attending physician had demonstrated that cotton placed in the right round window produced a striking improvement in hearing.

The patient is a slightly undernourished and intense young woman, extremely cooperative but deprecatory in regard to the importance of her personal problem. General physical examination was essentially negative.

Heart and lungs normal. Blood pressure 112/70. Pulse 80.

Otoscopic: Right—All of pars flaccida has disappeared. Part of the stapes may still be in position. The middle ear cavity is filled with granulation tissue. The round window niche is not completely filled. The surface of the granulations is moist but there is no frank pus. Left—There is a scar of a mastoid operation at the base of the ear. The floor of the canal is filled with a brown, tenacious material arising from a recess in the inferior part of the middle ear cavity. No landmarks can be made out. The rest of the cavity is dry.

Nose: The breathing space is clear. There is no congestion of the mucous membrane.

Tonsils are well removed.

Teeth are in good condition.

Sinuses are clear on transillumination.

Nasoscopic: The eustachian orifices are much restricted and distorted in shape.

Eyes: The pupils react to light and accommodation.

Reflexes are slightly hyperactive.

There is no general glandular enlargement. The thyroid isthmus is not palpable.

Patient has slight spontaneous nystagmus to left and marked past pointing. Romberg positive. Cranial nerves are negative except for the vestibular involvement noted above.

LABORATORY FINDINGS

Wasserman and Kahn: Negative.

Blood Examination: Hemoglobin 12 gm., 78%; R.B.C. 3,900,000; W.B.C. 6,100; polymorphonuclears 72%; lymphocytes 24%; monocytes 4%.

Urine: Negative.

X-ray: The right mastoid is normal, the left sclerotic. There is no active disease. The petrous ridges have a normal outline. The lateral of the skull is negative. The sinuses show no disease.

Culture from right ear: There are two colonies of Staphlococcus aureus. An attempt was made to sterilize this middle ear, first with sulfathiazol powder applied directly and finally with 1-1000 aqueous solution of Zephiran (Alba). Before attempting to place skin grafts on the granulations negative cultures were obtained.

Details of the procedures employed and changes in the patient's audiogram are shown in the accompanying chart (Chart III).

It will be noted that following the insertion of moist cotton, the placement of a rubber dam or a full thickness skin graft, the immediate improvement in hearing was quite remarkable. The patient's reaction to this improved hearing level after 23 years of a handicapping deafness was extreme. Ordinary street sounds, the rustling of leaves, corridor noises in the hospital were almost more than she could bear. As a result of this psychologic reaction it has been found advisable to maintain the hearing at a level slightly below the maximum improvement obtainable. Slight pressure upon the graft will cause an elevation in the hearing level of approximately ten decibels. The same shift has been observed following a change from the upright to the prone position. The only explanation of these changes would seem to involve either intracranial or intralabyrinthine pressure variation.

The patient no longer uses her hearing aid, and for all practical purposes has obtained complete acoustic rehabilitation. It is interesting to note that with the improvement thus obtained this patient went through various stages of elation and depression, at times feeling that her original impairment was preferable to the world of sound into which she has suddenly been injected. Though this effect of blocking the round window has been known, no detailed report of the phenomenon has been published. Further it is difficult to explain such a hearing level in any ear in the absence of the entire conductive mechanism.

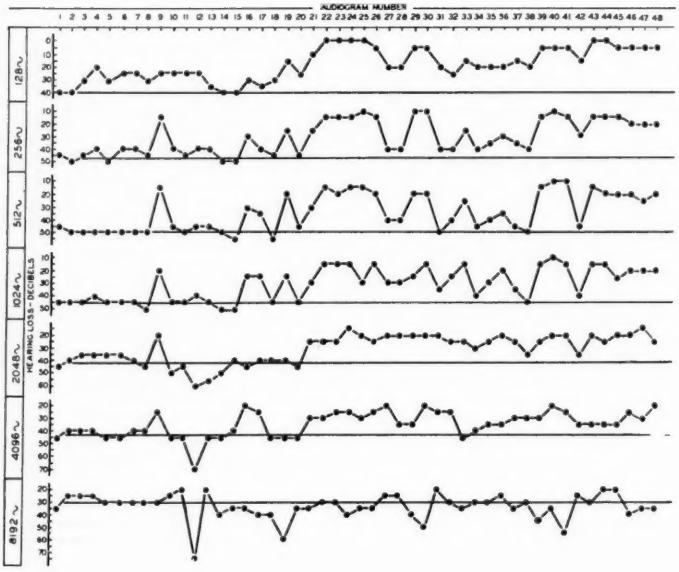

Chart III. This running audiogram of individual frequencies made over a period of approximately two months from September 23rd, 1940, to November 29th, 1940, illustrates the effect of blocking the round window niche in the chronically infected ear of the patient whose case history is given above. The solid black lines represent the base line of each frequency as determined in the control period (audiograms Nos. 1 to 8). No. 9, a cotton pledget moistened with Zephiran, which was subsequently removed, was packed in the niche. No. 12, the procedure was repeated without success, in fact with further impairment of the higher frequencies. From No. 16 to 20 further trials were made, the high points representing the successful effect of blocking the round window. No. 21, skin grafts were placed in the niche and over the granulating surface of the promontory. No. 27, the cotton pack and protective paraffin mesh were removed from the canal. Audiograms No. 28 and 29 illustrate the effect of upright and prone position respectively upon this patient's hearing, and with this evidence of a pressure factor a rubber dam was packed against the grafts at No. 30. These three audiograms were all done on November 12th. No. 31, the rubber was removed. This procedure was repeated at intervals from No. 32 to 43 when additional grafts were placed.

It has been shown experimentally that increased intralabyrinthine pressures tend to improve acuity and reduced pressures impair it.[23] Further studies were made using a purely mechanical method to modify intralabyrinthine pressure by changing cerebrospinal fluid, and the original observations were confirmed. These indicate that there is probably a critical level below which acuity is definitely impaired. A more direct method has been recently used in a series of experiments upon cats. A fluid reservoir of normal saline was connected with a manometer designed to register minus as well as positive pressure levels. This system was in turn connected with the subarachnoid space through a cistern puncture needle. By this method it was possible to raise or lower cerebrospinal fluid pressure to any desired level and maintain it at the level so chosen. Previous techniques[24] involved the changing of pressure levels by acute dislocation of blood vascular chemical changes either through the medium of hyper- or hypotonic saline solutions or specific drugs. There was always the possibility of direct effect of the chemical substance used upon the end organ. This mechanical method of pressure change precludes any possibility of such direct chemical action.

Again in confirmation of these experimental findings audiograms were made upon patients on whom spinal drainage was carried out. If the cerebrospinal fluid pressure in the lumbar space is reduced to zero, the hearing acuity gradually becomes impaired and returns to normal only after an interval of time sufficient for the elaboration of cerebrospinal fluid to restore the pressure to its original level (Chart IV).

EFFECT OF LUMBAR DRAINAGE UPON THE ACUITY OF HEARING

REPORT OF A CASE

The patient is a mechanic, aged 42 years. He complained of dizziness, tinnitus, and impaired hearing in the right ear. His family history is negative.

He had measles, whooping cough and diphtheria at the age of five and acute rheumatic fever at eight years. His general health has always been good, though he has been troubled with constipation and frequent head colds with postnasal catarrh. Tonsillectomy and adenoidectomy were performed in 1918.

He is a moderate smoker who seldom drinks. His present illness started three weeks ago with severe vertigo, nausea and vomiting. Marked tinnitus with impairment of hearing in right ear developed at the same time. He thinks he falls to the right and has been told that his eyes move in that direction.

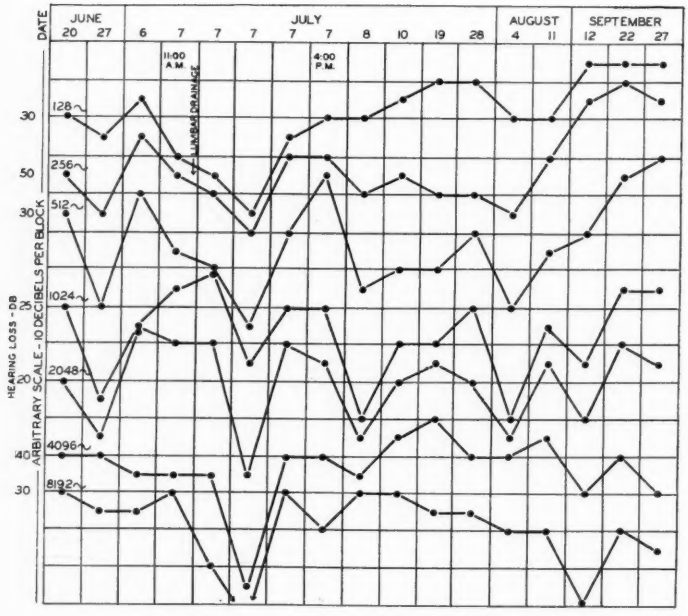

Chart IV. This running audiogram illustrates the effect of lumbar cerebrospinal fluid drainage upon auditory acuity. The patient also reacted to dietary and seasonal changes as indicated in the audiogram levels in August and September. Improvement in hearing was always associated with a reduction in the intensity of the tinnitus and freedom from vestibular symptoms. Variation or actual change in level from the control recording is indicated in the arbitrary scale of 10 decibels per block. Impairment is indicated by a lowering in the level of the charted line.

Otoscopic: The tympanic membranes were relatively clear; there was a slight atrophy with bulging of pars flaccida, right. The nose and throat examination was negative.

The general examination was essentially negative. Blood pressure 120/74, pulse 60. The heart and lungs were negative.

The audiogram shows fairly marked loss of hearing in the right ear both by bone and air conduction. Loudness balance test was variable in type indicating at least a partial neural involvement.

He was admitted to the hospital June 29th, 1939. The symptoms were controlled by a high salt diet.

LABORATORY EXAMINATIONS

X-ray: Negative.

Wassermann and Kahn: Negative.

Blood examination: Hemoglobin 15 gm., 97%; R.B.C. 5,160,000; W.B.C. 5,700; polymorphonuclears 61%;lymphocytes 31%; monocytes 1%; eosinophiles 7%; B.U.N. 15; ven. sugar 80; plasma chloride 580; plasma CO_2 61; ser. cholesterol 208; uric acid 3.2.

The patient had multiple small diffuse tumors scattered over arms and legs. Biopsy showed these to be fibro-lipomata.

During his hospital stay relief of symptoms was invariably associated with an increase in blood pressure and pulse rate.

On July 7th a lumbar drainage was done. Cerebrospinal fluid pressure was 170 mm. of water. Audiograms were made at intervals with pressure levels at 80 and 0 mm. of water. Hearing in the right ear was markedly reduced, but there was no change in the tinnitus. Hearing returned to its original level soon after the conclusion of the spinal drainage. Four hours later the general hearing was improved. Although no pressures were determined it is reasonable to assume that there was the usual rebound. To our knowledge no other observation of the kind has been reported. This patient has had frequent remissions and exacerbations of symptoms during the past eight months. However he has been able to continue his relatively hazardous occupation as a mechanic without interruption. Experimental findings relative to decrease in auditory acuity following reduction of cerebrospinal fluid pressure and its concomitant decrease of intralabyrinthine pressure have already been reported. Further clinical confirmation of these findings is important.

Modern concepts of body fluid pressure levels become more and more dependent upon seasonal, temperature, and barometric changes. Bazett[25] has shown experimentally that such atmospheric changes may profoundly affect the entire human organism. Body water balance and the resulting concentration of electrolytes, moderate changes in blood chemistry related to chloride concentration and CO_2 combining power affect every phase of normal physiologic function. Many acoustic and vestibular disorders are undoubtedly caused by these blood chemical changes.

Ménière's syndrome is the outstanding example of the effect. Treatment of this utterly disabling disease ranges from the two extremes, Dandy's[26] section of the vestibular division of the nervus acousticus and Furstenberg's[27] dietary regime which in many instances is almost worse than the disease itself. Dohlman[28] demonstrated a possible role of the vestibular cupola in these paroxysmal seizures. His work is completely convincing from an anatomic standpoint but offers little if anything to therapeusis. However, the recognition of seasonal influence with its profound body fluid

changes lends support to the control of electrolytic balance in the treatment of this complex. It has been possible purely on the basis of sodium chloride intake and fluid output to control vestibular seizures and restore patients so afflicted to normal activity. Hallpike[29] has reported the autopsy findings of two patients operated upon by Cairnes for Ménière's disease. Sections of the temporal bones showed marked distension of the cochlear duct, as evidenced by a bizarre displacement into the scala vestibuli of Reissner's membrane. In these two cases the unaffected side was used as the control. In many experimental animal serial sections such bilateral irregularities have been found due to errors in histologic technique. Hallpike's cases were prepared for fixation several hours postmortem after the most favorable time for histologic preparation. Before accepting these observations as final much positive confirmation must be available.

These patients exhibit gastro-intestinal and biliary disorders with considerable frequency. But here again there is a primary derangement of the NaCl and CO_2 balance in the blood. Ménière's disease is characterized by remissions and exacerbations. Any medical treatment must be subject to this seasonal change. Patients have been seen who exhibit profound symptoms or complete relief in direct relationship to their geographic habitus during the various yearly atmospheric changes. Body fluid changes can, in some cases, be reproduced by control of NaCl intake and output, and hearing impairment, vestibular disorders, and tinnitus can be controlled or precipitated by the management of the chloride intake. It is felt that the electrolyte is of more significance than the sodium ion of the radical.

ENDOCRINOLOGY

It is probably incorrect to associate endocrine deficiency or hyperfunction directly with disorders of hearing. As the total organism is affected by the adenopathy, the acoustic mechanism may reflect the general asthenic state of its host. By replacement or substitution it is possible to improve the general constitutional level to such an extent that all the special senses may exhibit some benefit from the particular therapy. It is felt that aside from frank hypertensive old age deafness, impaired hearing is associated with low normal blood pressure levels. These individuals are essentially below par, are more adversely subject to fatigue, cold, heat, respiratory diseases and all the usual health hazards of normal living. By replacement therapy they acquire certain resistance and immunities which may well rebuff the attacks of common epidemics so often

associated with the development of hearing loss. A case in point, a young musician with a B.M.R. of —25, is completely dependent upon direct replacement therapy and avoidance of repeated upper respiratory infection previously associated with his specific glandular lack for continuance of well being and a reasonable support of his hearing level.

Probably of the greatest interest in this particular phase of the situation is the adren-ovarian-pituitary complex with its complicated menopausal implication. The entire acoustic and vestibular apparatus may be involved. Hearing impairment, vertigo, and tinnitus may be outstanding difficulties regardless of blood pressure changes so often associated with the climacteric. The anatomic manifestations are many. In the pituitary group changes in the nasopharyngeal mucous membrane and hyperostosis of the frontal bone are anatomic facts. Psychic reactions, extremes of emotionalism with instability of temperament all enhance a hearing loss which may or may not be of real significance. In this group many important results may be obtained by the use of certain estrogenic substances. Without the authority or even the necessity of actual statistics this group of patients makes up a very large percentage of the otologist's practice.

However much the prefix "pseudo" may be abhorred in connection with any disease entity it would be difficult to bar it from some relationship with Ménière's diesase. The symptomatology of the disease is specific. Specific surgical treatment in intractable cases is curative, but many less clear cut cases respond to some form of medical therapy. May not the distinction between pseudo and frank Ménière's disease be based on the final necessity of surgery? If this standard be accepted, surgery should be resorted to only under the most extreme necessity.

In this direct connection attention must be drawn to the importance of a psychologic appraisal of every person presenting a hearing impairment. Depression is the usual result. Only one important otologist records an insistance upon routine psychologic studies of his patients (Canfield[30]). It should be a matter of routine for everyone concerned with the treatment of deafness, but few otologists are qualified to carry out such studies, and it is doubtful if many psychologists are fitted to appraise mental states in relation to hearing loss. The literature is peculiarly devoid of data relative to this very important subject. There are two aspects of the problem. First is the psychologic effect of deafness with all its intro-

versions and withdrawals from social contacts. Second, and unfortunately far too frequent, is the extraordinary hysteric reaction to occasional relief of deafness. When the otologist becomes truly interested in this phase of the problem or when he can enlist the interest of the psychologist, much will have been accomplished. The otologist's "sympathetic approach" is not enough. A complete psychologic appraisal is essential.

VITAMINS

There is no more completely disappointing phase of the therapy of deafness than the supposed effect of vitamins upon hearing. A critical analysis of the effect of vitamin therapy in human deafness begets a feeling of complete futility. From an anatomic standpoint alone, neural impairment of hearing is final and in its manifest and individual degree, complete. Why should Vitamin B or any one of its various complexes restore function to an organ of Corti deprived of its neural mechanism? The sooner this misrepresentation is corrected the sooner the reputation for honesty of otologists and the peace of mind of the deafened individual will be achieved. Controlled vitamin experimentation in relation to deafness presents almost insurmountable difficulties. Only extreme anatomic effects are demonstrable experimentally. The intermediate phases of hearing loss, if and when they occur, are not a matter of record. It is most unlikely that any of the neurologic effects produced by extreme experimental lacks are significant from the standpoint of human clinical deafness. All reported results of therapy are completely inconclusive.[31, 32] There is no anatomic evidence that the anatomic result of dietary defects is corrected by appropriate vitamin replacement. At the moment it would seem that this entire subject is lacking in true scientific control which is possible only with animal experimentation. Any human audiograms, so far presented, are of no real significance if taken as a measure of the effectiveness of vitamin therapy.

VACCINE THERAPY

Every otologist has prescribed vaccines of one commercial kind or another with varying degrees of success. However, controlled groups, particularly those in various colleges, east and west, have shown that available "cold vaccines" are of no real importance as true preventive measures. Aside from studies of large groups so treated every otologist has observed acute reactions to vaccine therapy which definitely jeopardizes the integrity of hearing. From the

purely otologic standpoint vaccine therapy cannot be considered a matter of any real consequence.

DRUGS

For many years certain drugs have been associated both in the lay and professional mind with the development of deafness. Certainly tinnitus following the use of salicylates and quinine is an established fact. Mosher[33] has shown experimentally that the ingestion of quinine produces hemorrhages in the inner ear of guinea pigs. Taylor[34] for years has been crusading against the use of quinine in obstetrical practice, on the theory that it may be responsible in some measure for the incidence of unexplained congenital deafness. Direct evidence of a causal relationship, however, is lacking. A survey (unpublished) of families of pupils in one of the country's largest schools for the deaf failed to reveal any association between the use of drugs by the mother before, during, or after the pregnancy resulting in a biologically deafened child. The matter was gone into with extreme thoroughness even to the use of drugs as contraceptive agents. Despite this negative clinical evidence there is no question that all suspected drugs should be completely avoided during pregnancy. Our ignorance of this entire subject of biologic deafness is so extreme that no safeguards against its development should be neglected whether or not actual supportive evidence is available.

Again most otologists accept the theory that the use of drugs may contribute to the development of adult deafness. Certainly such a causal relationship in ordinary practice is not a frequent one, and when found, the hearing impairment is often not the type expected. Probably alcohol and tobacco are more generally used than any other supposedly toxic agents, and yet their association with deafness is certainly not a striking one. The therapy of deafness by the use of drugs is on no more secure a basis than that of vitamins. The much heralded thyroxin treatment of otosclerosis, supported by Goldstein,[35] failed completely to live up to its original promise and has been abandoned. More recently prostigmine has been reported[36] as effective in the treatment of both deafness and tinnitus. Houser[37] and others have been unable to confirm the original observations.

An experimental and clinical study[24] of the direct effect of ephedrine, benzedrine and mescaline upon the acuity of hearing failed to establish any specific actions of these drugs upon the ear. Any effect obtained was probably due to changes in blood and cere-

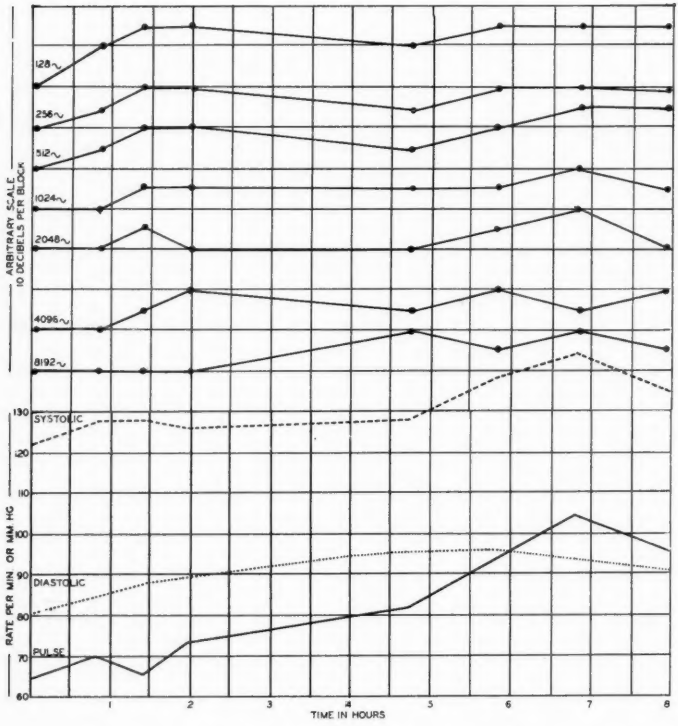

Chart V. Composite audiogram and record of the blood pressure and the pulse rate of a young adult with normal hearing after the oral administration of 10 mg. of benzedrine sulfate. A 10 to 15 decibel improvement in hearing occurred for all frequencies except 4096 cycles at the height of the change in systolic pressure. In this chart the initial level of hearing for the different frequencies is not indicated. Variation or actual change in level from the control recording is indicated in the arbitrary scale of 10 decibels per block. Improvement is indicated by an elevation in the charted line. (Reprinted from Archives of Otolaryngology, 29:903 (June), 1939).

brospinal fluid pressure and was relatively fleeting in character. A case already reported which demonstrates this clinical improvement is shown in Chart V.

SURGICAL TREATMENT

The present critical interest among American otologists is directed almost completely towards the dramatic operative procedure of fistulization of the horizontal semicircular canal for the relief of otosclerotic deafness. Of distinguished heritage, this re-

markable procedure has been recorded in detail too often to need recapitulation here. Its modern exponents are the Frenchman, Sourdilles of Nantes, the distinguished Holmgren of Sweden, and his great pathologic collaborator, Nager of Zurich. The whole technique has been adapted and elaborated with consummate finesse by Lempert[38] of New York. In 1930 the present author read Sourdilles' original and unpublished manuscript in Paris describing his elaborate operative technique which involved repeated major surgical procedures upon the affected ear. No critical studies of hearing loss and postoperative improvement were presented then, nor have they been since.

Holmgren's[39] painstaking and authoritative efforts, all of which have been presented in detail, must be regarded as the true basis for this approach to the surgical treatment of deafness. Details of the operative technique are a matter of record in America, and the operation is being performed in practically every important otological clinic in the country. Unfortunately improvement resulting from the long and complicated operative procedure with its attendant hazards has not proved a lasting one in all cases. Lempert reports permanent improvement in 126 out of 150 operative cases, and yet the oldest of his series is well under the proposed standard of five years.[40] The fistula closes as a result of normal bone regeneration, and the improvement in hearing is lost. So-called "revision" or reoperation is then necessary, and again from known activity of bone growth the fistula may close. No effort beyond the trial and error method has been attempted to study the actual anatomy and physiology of this particular phenomenon except by Canfield[41] in this country. Continuing his studies begun with Holmgren, Canfield has attempted experimentally to determine factors which might maintain the patency of this minute opening in the horizontal canal and so assure the actual improvement in hearing achieved by the relatively empiric procedure.

Suggestion that the actual physiology of the operative effect should be determined has met with the retort that such knowledge is unnecessary so long as the clinical results are definite. This seems hardly a proper or reasonable attitude. Fenestration provides a new window, and by so doing, of necessity decompresses the labyrinth. The perilymphatic sac is opened, but the endolymphatic sac should be preserved intact. However a brilliant immediate effect was obtained by Lempert when the endolymphatic sac was opened in the course of one operation. If the fenestration of a canal is so effective why could not the same result be obtained by fenestration carried

out in the much more accessible promontory? Holmgren did perform this operation and obtained improvement but abandoned the procedure due to the fact that in the vestibule the endolymphatic sac is adherent to the bony wall of the promontory.

The insistence that the operation be performed only in the presence of true otosclerotic deafness with normal bone conduction gives rise to several important questions. Otosclerosis is a disease with definite pathologic manifestations. The final proof of its presence is microscopic examination of the inner ear itself. Pathologic evidence and reported clinical incidence are too widely divergent, the former with a ten per cent incidence, the latter eighty-five per cent. We must conclude that correct diagnosis of this condition can be made in only a small percentage of suspected cases. It is known on the basis of the recoupment theory that neural impairment may exist and that with sufficient amplification of the acoustic stimulus the remaining intact neural mechanism will function in a satisfactory manner. Further it has been shown that bone conduction response is not necessarily a measure of a completely normal neural mechanism.[42] Improvement in the functional testing of bone conduction has been neglected in the general desire to achieve accurate measurement of air conduction acuity. The most important research in this particular phase of the situation is that reported in the fundamentally important monograph by Barany.[43]

Finally in the fenestration operation a palliative procedure is carried out. The symptom, "deafness", is corrected with more or less success. Nager[44] has shown that the otosclerotic change is progressive and in advanced cases may extend from the actual confines of the capsule and actually involve the scalae, thus obliterating those minute fluid spaces and ending finally any possibility of auditory response from the affected ear. However the interlude of improved hearing, whatever its duration, might well be considered compensation for the operative procedure.

In this laboratory a considerable number of animal experiments have been made to determine the acute effect of fistulization at various points on the cochlea from the promontory to the apex. The effect upon auditory acuity was measured by the electrical potential change of the cochlea itself and also of the auditory nerve. In addition a comparison was made between threshold response and an established optimum intensity level of acoustic stimulation. This latter approach was used in an attempt to answer the frequent objection to threshold audiograms as a true measure of hearing. This

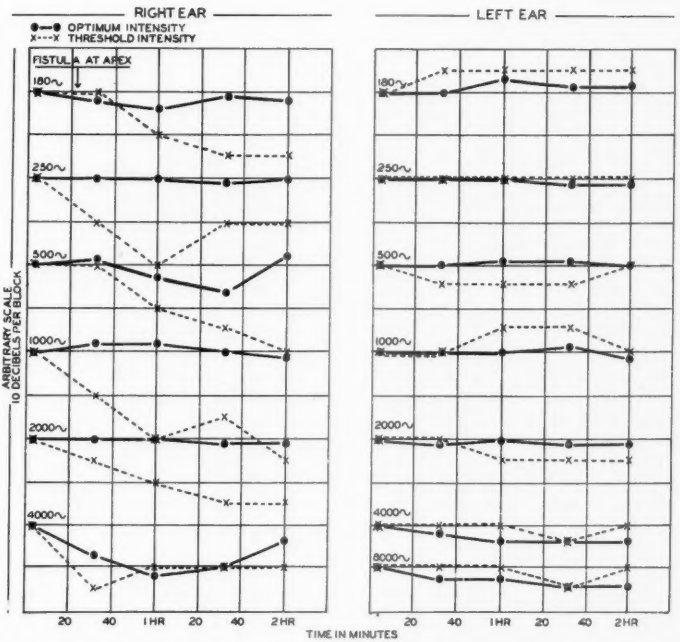

Chart VI. An apical fistula made in the cochlea of a cat's ear illustrates the effect upon threshold and optimum intensity. During the time of this experimental observation, two hours, there was a progressive falling off of the threshold level but no reduction at optimum intensity. The left or control ear remained at a constant level for both intensities of stimulation throughout the experiment.

point has been brought up repeatedly in the past few years in an effort to reconcile subjective improvement with complete lack of audiometric evidence. Chart VI illustrates the results of one experiment of this type on a cat.

Under light ether anesthesia both bullae and middle ears were opened and both drums found to be clear and delicate. A cistern puncture was made, and the cerebrospinal fluid pressure was 100 mm. of water. Electrodes were placed in both auditory nerves through trephines made over the lateral cerebellar lobes, with the ground electrode in the back of the neck. Measurements of electrical potential changes in the auditory nerves following acoustic stimulation of the ears were made at threshold and at an optimum intensity on

both ears. As in all previously reported experimental findings this optimum or standard intensity of acoustic stimulation is that which will give a relatively uniform response under practically all experimental conditions. The chosen intensity, varying as it must for individual frequencies, would correspond in general to the level of normal speech intensity, a level lying between threshold audibility and a point where over-load would develop. For simplicity, all subsequent tests have been referred to this first test and charted accordingly.

Following these control tests a small fistula was made at the apex of the right cochlea with no damage to any of the intracochlear structures and no hemorrhage. Fairly free drainage occurred throughout the remainder of the experiment, tests being made at approximately twenty minute intervals over a period of two hours. Note that the response at optimum intensity is unaffected by the apical fistula whereas the threshold intensity level is markedly impaired. In the left or control ear neither optimum intensity response nor threshold have changed during the two hour period of observation. A final cistern puncture showed the cerebrospinal fluid pressure to be 130 mm. of water. This is the first experimental demonstration of a dislocation between normal intensity of hearing and threshold.

TINNITUS

This very important affection of the ear has been much neglected both by experimental and clinical investigation. The reason is obvious. Little is known of the true cause of tinnitus, certainly of the inner ear or central types. In general it defies all effort towards alleviation, and yet it is the most harrassing of all otologic conditions. Of itself it can produce a disabling hearing impairment by the simple effect of masking true auditory acuity. It is entirely possible to measure the intensity and determine the frequency of any single frequency tinnitus (Fowler[45]). Proper understanding and rational therapy of these head noises offers a commanding challenge to every investigator in this field. It is possible to produce a pulsation tinnitus experimentally, one which can be demonstrated by the electrical method of cochlear or nerve response, but no individual frequency tinnitus has been observed as either a spontaneous or induced phenomenon in animals in this laboratory.

As has already been stated relative to drug, vitamin and endocrine therapy of deafness, nothing of significance or real impor-

tance has been presented in the treatment of tinnitus up to the present time.

DIAGNOSTIC METHODS

During the past decade the diagnosis of deafness has passed from the period when the otologist confirmed the patient's opinion that his hearing was impaired to the present time when instruments of complete precision, such as audiometers, are used to gain a definite knowledge of the individual's threshold hearing level. The committee of the American Medical Association appointed to establish standard requirements for these instruments in collaboration with the Government Bureau of Standards, has finally reported the necessary criteria of acceptance. Several commercial audiometers have been accepted. Intelligent interpretation of these audiograms is, unfortunately, not a matter so easily controlled. Papers have appeared, "The Selection and Operation of Audiometers"[46] "The Inherent Accuracy of Repeated Clinical Audiograms",[47] but no adequate handbook on the use of these important instruments is available other than those compiled by individual manufacturers. It is hoped that some such volume will soon be available for otologists who are willing to make this very considerable investment in diagnostic equipment.

A recent survey of a random group of audiometers all in daily use reveals several interesting facts. Despite standardization, comparison of audiograms between audiometers is relatively unreliable even though repeated audiograms on any one instrument may be accepted within definite limits of error. The often discussed checking of individual instruments is still much neglected. In this connection the manufacturers are as much at fault as the otologists. Every maker should sponsor such periodic checks either by inspection in the physician's office or by calling in the instrument for examination in his own laboratory. Much dissatisfaction would thus be avoided.

MASKING

Few masking devices now available can be relied upon either in regard to the calibration of intensity or the frequency range of the masking noise. Here again the otologist's lack of familiarity with essential requirements and rules of application of masking are largely at fault. Too much emphasis cannot be laid upon this all important diagnostic method. Following rules laid down,[40] only

with masking is it possible to show that air or bone conduction is completely absent in unilateral deafness. The extreme importance of these findings may prove the differential factor between palliative and direct surgical approach. The properly equipped otologist should decide whether or not brain surgery should be undertaken for the relief of acoustic pathology. Neuro-surgeons are peculiarly hesitant to operate upon these cases. Exploration of the cerebello-pontine angle is a relatively benign procedure. The removal of an advanced angle or acoustic tumor is far from benign and seldom successful. So soon as audiometers have attained uniform and reliable excellence this important feature must be given proper consideration, and masking devices will be used with real assurance as to efficacy under all circumstances.

LOUDNESS BALANCE

One of the most important diagnostic procedures available at the present time is the "Loudness Balance Test". This represents a splendidly scientific research effort on the part of Steinberg and Gardner[48] and its clinical application by Fowler.[49] The theoretical basis of the test has been supported by the anatomic studies of Lorente de Nó[50] The test itself is the only absolute differential method in the determination between conductive and perceptive deafness. Where bone conduction findings are equivocal, the "Loudness Balance Test" will invariably resolve the difficulty. There is no need to detail the technique of the procedure here other than to say it is entirely practical, is not a burdensome test for any patient and not unduly time consuming for the examiner. It should and undoubtedly will receive much more general application when its advantages and significance are more generally appreciated and the otologist is willing to procure the additional equipment necessary. One eminent otologist refuses to subject his patients to this test and yet persists in the inflation and bougienage technique.

FATIGUE TEST

A direct application of experimental findings[51] is that "clinical fatigue" of the ear is another differential factor between conductive and perceptive deafness. Under all reasonable intensities of sound stimulation an ear impaired by involvement of the conductive mechanism will not fatigue whereas in a case of perceptive deafness, fatigue will develop if the ear is subjected to a frequency of high intensity for periods of time ranging from five minutes to ten minutes. Sound sources capable of producing intensities 80 to 90 decibels

above threshold must be available for the proper performance of this test.

These diagnostic procedures have been discussed in some detail, for without such accurate audiometric study and an intelligent knowledge of the constitutional and psychologic background in each individual case, therapy can hardly be of any real importance.

PREVENTION AND ALLEVIATION

Though prevention and alleviation of deafness should properly come first in any such discussion as this present one, its importance from the standpoint of original contribution is unhappily insignificant. As has been stated before, control of acute infections of the ear, proper handling of acute mastoiditis, prevention of complications in acute upper respiratory and contagious diseases, control of scarlet fever, the virtual elimination of diphtheria, have all undoubtedly reduced the incidence of deafness in children. However these cannot be claimed as contributions of otological investigation.

Birth control in marriages where hereditary taints appear in the history of either partner may prevent its continuance in an occasional offspring, but it will also deprive many a normal hearing child of existence. Sterilization as practiced in certain European countries is hardly warranted. In a survey of families of biologically deafened children there is no immediately apparent relationship between age or health condition of the parents, order of pregnancy, mother's health or occupation during pregnancy, ease or difficulty of labor, neo-natal or late post-natal care. The truly congenital deaf have already been mentioned and discussed.

Crowe[52] has reported observations upon a large group of adolescent children in whom adenoid tissue in the eustachian orifices has either been observed or surmised on the basis of high frequency hearing loss. During this age period the growth of adenoid tissue can be controlled by direct application to the orifice of radon emanations. As in the less direct and probably less effective X-ray treatment of the eustachian orifice and the tubes there occur apparent seasonal exacerbations following the remissions, and the treatment must be repeated. By both methods striking improvement occurs in favorable cases. Crowe feels that with such radiation in proven cases of adenoid involvement and its empiric use in suspected cases, fifty per cent of human deafness will be eliminated. Actual realization of such a thesis would indeed be a magnificent accomplishment.

DEGREE OF IMPAIRMENT, DURATION AND AGE

It is now generally recognized by the more conservative exponents of any form of treatment, whether medical or surgical, that three important factors mark the dividing line between probable success or failure of any therapy, namely: age, extent of impairment and its duration. It would be difficult to differentiate in extreme instances between the relative importance of these factors. In the natural course of events the three would be concomitant. But certainly, regardless of duration or degree, the development of a hearing loss in the fifth decade and upward is essentially less amenable to any form of therapy than in the younger age groups.

Duration, if a matter of years, carries with it the additional factor of speech involvement, actual loss of auditory memory and all the implications of these critical factors. The degree of impairment in young or old at the outset limits the possibility of improvement. In no reported case, except that mentioned earlier in this paper, has a consistent improvement of more than 20 to 30 decibels in the hearing level throughout the critical frequency range been achieved. Quite obviously the original level must be such that an improvement of this extent will be practically effective. This critical impairment level is generally accepted as 50 decibels of hearing loss in the frequency range 256 to 4096 cycles.

A final phase of this whole complicated situation is the problem of therapy in relation to the factors just detailed. Patients come to a specialist, in the present instance an otologist, expecting, even demanding, treatment of almost any kind. Is the physician warranted in carrying out a course of therapy which he knows has slight chance of success? Few patients have acquired a philosophy which will permit them to accept the frank declaration that they must make the most of their handicap. They will continue their almost endless round hoping that eventually the miracle of relief may be achieved. The problem is more an ethical than a therapeutic one and permits of no ready solution. But if a patient's confidence can really be gained by the otologist, much will be achieved in the solution of a difficult problem.

Two important groups present themselves for the otologist's consideration. For convenience they will be classified as young and old. The former extends through the fifth decade; biologic deafness is not considered here. In the older group all deafness manifests itself in a relatively gradual manner from the age of 50 onward. Therapy can be legitimately applied with, however, scant hope of

benefit, in the younger group. Except for the treatment of chronic upper respiratory infection, sinus disease, and the constitutional defects of whatever kind, attempts to correct hearing losses in the older group are almost uniformly futile. In both groups there are the three important categories of hearing loss, "Impairment", "Hard of Hearing", the euphemism of the deaf, and the "Deaf". The first of these may respond in some measure to our best therapeutic effort. The third responds to nothing. The otologist's greatest contribution here is to convince these relatively unhappy people that routine therapy is both a waste of time, theirs and the otologist's, and particularly a waste of their own money. Frequently requests are made for audiograms on patients in the age group 50 to 60 years who are having bi-weekly eustachian inflations and dilatations by otologists whose responsibility should be to convince these patients that no local treatment, allopathic, homeopathic, or chiropractic, can by any possible chance warrant their subjection to it.

HEARING AIDS

Not as an admission of defeat but as a tribute to acoustic engineering must the otologist accept the completely important contribution to the alleviation of deafness, the electrical hearing aid. No other effort, certainly none on the part of otologists, has contributed so much to the immediate relief of deafness, particularly in the older age group, as has the modern hearing aid. Realizing our own futility in regard to routine treatment we should urge every one of our patients in this particular group to use such a device. This is not easy of accomplishment. Neither men nor women are willing to admit a defect which is not immediately apparent. In the case of the individual who is willing to use an appropriately fitted hearing aid, the otologist must assume a serious responsibility.

Bone conduction aids on account of their relative inconspicuousness have been exploited and are preferred by most deafened individuals. In general it may be said that no one over 50 can achieve more by bone conduction than air conduction, if as much.

The whole question of prescribing hearing aids is in a complete state of flux. The reason for this situation is quite obvious. Prescription with its attendant compensation for specific hearing loss is not a matter of record at the moment. Compensation for high or low frequency loss is invariably achieved at the expense of some other factor. We do not feel that compensation, per se, is of great consequence. All audiograms are different. The individual's preference for amplification of high or low frequency is unpredictable.

Different hearing losses involve different compensations. The practice in this laboratory has been to measure on a percentage basis, speech intelligibility. Hearing aids have been made available for these tests through the courtesy of various manufacturers. If an equally high percentage of achievement is obtained with two or more aids the final selection is left to the patient on the basis of preferred quality of sound reproduction. This is a time consuming procedure and is fraught with difficulty. There is apparently no solution to the hearing difficulty of the patient 60 years or over with a sharp drop at 1024 or 2048 cycles, the typical neural deafness of advanced age. The claim of manufacturers that their particular hearing aid can compensate for specific lacks in hearing is not now and probably will never be a matter of actual achievement. There is no electrical system now available nor any system of filters which can resolve a discrepancy of 50 to 70 decibels loss in the high frequency range with a relatively normal low frequency level. With some hesitation the suggestion is offered that the otologist dampen in some way the low frequencies in these ears so that the hearing loss can be compensated for by an aid of a relatively flat characteristic frequency curve. We have yet to see any modern hearing aid of the so-called "fitted" variety which compensates adequately for this type of hearing loss.

But it is necessary to go back of this immediate problem. Much has been spoken and written relative to the "prescription" of hearing aids. There is actually no analogy between the prescription of hearing aids and eyeglasses. Ophthalmology and optometry are special fields requiring specific training in a field of exact science. An otologist is an individual trained in the handling of acute and subacute diseases of the ear. Neglect of such immediate difficulties results in complications which involve definite hazard of life. Hearing loss is incidental. Few otologic chiefs of staff are trained or even have knowledge of the acoustic problems involved in specific types of deafness. It is obviously quite futile to expect a practicing otologist to be concerned with this very thankless task of prescribing hearing aids.

If this fact is accepted, and at the moment it must be, there is no alternative to the exploitation of hearing aid manufacturer's audiometrists. A recent advertisement by one of the better hearing aid companies pictures such a test being conducted by a rather distinguished looking middle aged man. The patient signals brightly in response with an upraised finger but without any visible connection via ear phone with the audiometer. Such flagrancies of adver-

tising techniques are not conducive to a feeling of confidence in the product offered for sale. We can only hope that men trained in the modern methods of medicine may be willing to devote time and effort to this very important new aspect of the general hearing problem.

This particular point leads to the special difficulty of the hearing aid problem and is a factor over which the otologist has no possible control. A hearing aid is found suitable and recommended. The cost of better instruments can be set at $150.00, slightly more or less. Few patients can afford to make this outlay whether it involves rehabilitation or not. One of these companies offers what amounts to a "second hand" service. They are to be complimented. More affluent patients turn in to the otologist their older models, but there is no means whereby they can be reconditioned for the less fortunate individuals. We would suggest some such "servicing" system for every make of hearing aid, sponsored by representative otologic societies with branches in every important city. These societies already sponsor research programs of every sort and importance. No more immediately practical purpose could be served than by this one suggested, and every recognition is accorded in this connection to the efforts of the various leagues and societies organized for the alleviation of the problem of deafness.

DEAF SCHOOLS AND METHODS OF TEACHING, ORAL AND AURICULAR TRAINING

With the advent of radio and its application to acoustic problems a new era immediately developed in the pedagogic methods for the education of deaf children. Today in many schools for the deaf one-third or more of the pupils enrolled are being educated by a combination of oral and auricular methods. It has been estimated that the total period of training for this fortunate group will be reduced by at least two years. This is a matter not only of social but economic importance. No real evidence is available that hearing is actually improved by auricular training despite many statements to this effect. However, voice training and intelligible speech are immeasurably superior as a result of the auricular method.

It is now felt that all deafened children regardless of their actual audiograms should be given an opportunity to use group hearing aids and the method abandoned only after a trial period of sufficient length to prove it ineffective. At the Pennsylvania School for the Deaf, specially designed group hearing aids of particularly high

quality and unusual power are being used in an effort to reach pupils with profound hearing losses. It is now an established fact that these deafened children require a period of at least two years to be taught to hear regardless of the level of their residual hearing. For this reason the youngest pupils, six to eight years of age, are to be taught by the auricular method as a matter of routine.

At the Pennsylvania school all children are examined audiometrically once or twice each year. Voice recordings are made of each child once or twice each year. This material is obtained by a resident otolaryngologist who has a full time position at the school. A recent study has been made of the relationship between patency of the eustachian tube and hearing loss upon practically the entire enrollment of the school, a student population of 540.[53] Studies have also been made in collaboration with the Bureau of Child Hygiene in Washington relative to hearing loss et cetera in this group of children.[3, 54]

SUMMARY

From the material presented in this survey one very striking point becomes evident. Interest in the problem of deafness has increased markedly during the past ten years and activity quickened in many fields of investigation. The physiology of hearing has been established upon a rational and fundamental basis. Old outmoded forms of therapy are gradually being discarded, and new methods have been developed as a direct result of knowledge gained in experimental laboratories. The surgery of deafness will undoubtedly progress very rapidly in the next four years. Endocrine, vitamin and drug therapy, though at the moment exploited far beyond their proven value, offer fascinating problems for research both in the matter of prevention and alleviation of deafness. Their ultimate place in otology will certainly be one of very great importance.

The development of new and exact diagnostic methods has progressed during this period probably more than any other related factor. Here, again, knowledge gained in experimental laboratories has found direct application in clinical otological practice. This has been due very largely to the rapid advances made in radio engineering during this past decade. Research carried out in commercial laboratories has made audiometers and accessory equipment, instruments of precision and within the means of practically every otologist. The appraisal of a hearing loss is now a matter of exact record and not merely one of opinion.

Hearing aids will soon be equally precise and standardized. Their rapid development has been truly remarkable. When stabilized, the change from carbon microphone to the newly developed vacuum tube sets will bring immeasurable help and comfort to many seriously handicapped people. The only drawback at the present is the extreme cost of all the better instruments.

In conclusion it would seem that the future promises much in the form of real help in the treatment of this handicapped group.

ABINGTON MEMORIAL HOSPITAL.

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X

THE WALTZING (CIRCLING) GUINEA PIG*

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The waltzing guinea pig is a peculiar strain of guinea pig first found by Professor Ibsen of Kansas State Agricultural College. A report of this peculiar animal was made by Ibsen and Risty[3] in December 1929, and was followed later in 1932 by a report of Professor Ibsen[4] on modifying factors in guinea pigs. These interesting animals were sent for study by Professor Ibsen in 1933. A preliminary report was made on these animals in 1939 to the American Otological Society.[7, 8]

The waltzing guinea pig not only tends to run in circles but is also deaf. The circling begins very soon after birth and becomes pronounced on the third to fifth day. There is also with this circling of the young animal a tendency to throw the head backward and a trembling or tremor of the head. The animals do not respond to external sounds of any kind except when they feel the vibration as a result of movement of air or of the cage. There is no pinna reflex to sound, and the animal acts unconcerned in the presence of loud explosive sounds. The normal guinea pig gives a definite pinna reflex to sound and becomes very much excited.

The circling can be in either direction and the animal, when excited, will whirl for considerable periods of time. The circling is not due to being confined in a cage, for when they are placed in a large open space the circling still continues. They can run for short distances in a straight line but soon begin to circle. This circling is very pronounced when the animal is hungry and also occurs at periodical times during the day. The attendant reports that early in the morning they are very excited and circle with great intensity. This may be due chiefly to hunger or the excitement caused by the presence of the attendant.

The animals grow very slowly. The six months old waltzing guinea pig is no larger than the two-to-three months old normal

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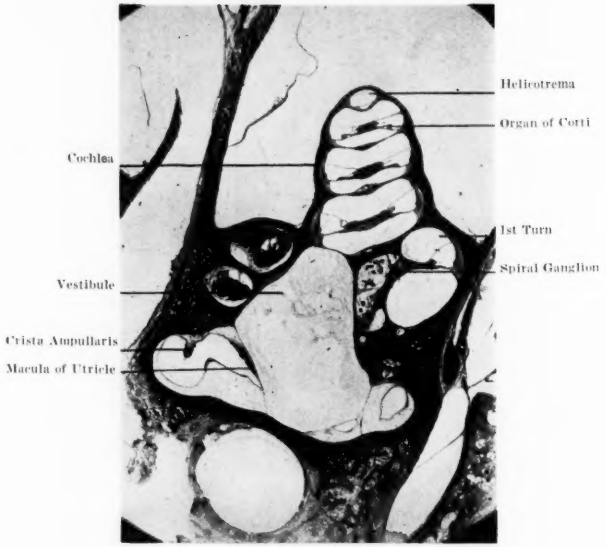


Fig. 1. Vestibule and cochlea X15. Waltzing Guina Pig. Section of cochlea shows 4 turns. Section misses the modiolus. Note the different degrees of degeneration of the organ of Corti. Almost normal appearance of the spiral ganglia of the first turn. Vestibule shows macula of utricle and the crista ampullaris of a semicircular canal. Utricle and crista ampullaris are normal.

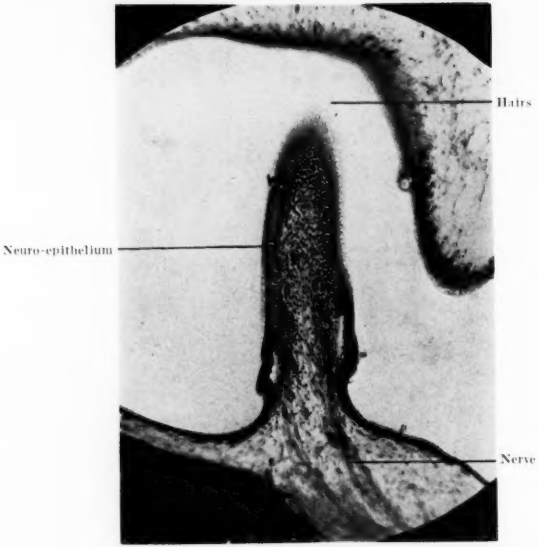


Fig. 2. Crista Ampullaris X260. Waltzing Guinea Pig. Tangent section through the crista. Showing normal neuro-epithelium and nerve supply. The hairs can be made out extending into the cupola.

guinea pig. The circling and deafness of these animals persist throughout the life of the animals. Animals now four to five years of age are still circling and show all the characteristics present at birth.

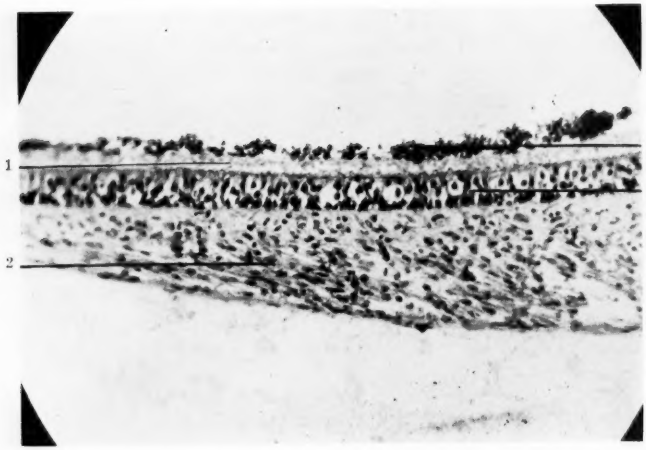
The inheritance of the deafness and circling is a simple Mendelian recessive characteristic. There is also some evidence for the presence of a semi-lethal factor as the animals are very difficult to raise when waltzers are bred together. A number of the offspring die within the first two weeks of life or are still-born. In the breeding of these animals it has been found necessary to breed the waltzer with normal animals and then rebreed the hybrids with waltzers to reproduce the strain. All hybrids were found to be normal for hearing and vestibular reactions.

The circling varies with the animals, some moving in large circles slowly, others moving very rapidly in small circles. They will circle around another animal or object placed in the space.

The head of the animal when circling is slightly tilted, the side nearest to the circumference of the circle being higher than the one nearest to the center. When running in a straight line there is a peculiar swinging gait with the head weaving from side to side and being slightly tilted. The circling will be in either direction, and the animal may reverse itself, though it will continue for long periods of time in one direction.

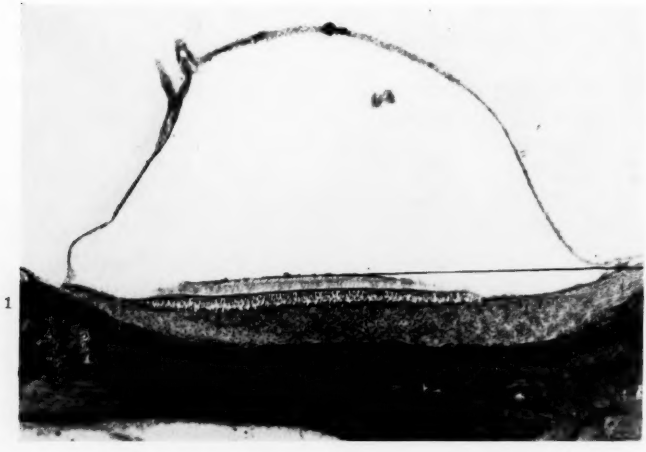
The eyes show no nystagmus during circling or on movement of the head from side to side. Neither is there nystagmus on moving the animal from side to side. The normal animal has eye movements as a result of such motions. Cogan[2] has shown that these eye movements of the normal animal are the result of vestibular stimulation. The lack of these eye movements is to be expected in the waltzer, for if present as a result of the vestibular stimulation, it would be impossible for the animal to continue its existence. There must be one of two conditions present in these animals: 1) An abnormal or absent vestibular apparatus, degeneration of the macula or crista or degeneration of the vestibular nerve, 2) a normal vestibular apparatus and vestibular nerve with a central nervous break of the neural connections with the ocular motor nuclei and postural nuclei of the midbrain and medulla.

The righting reflexes of these animals on casual examination appear normal. The righting reflexes were studied in detail by Cogan.[2] He found that these animals right themselves chiefly by



1. Hairs of neuro-epithelium.
2. Nerve to macula.
3. Neuro-epithelium.
4. Otoliths.

Fig. 3. Macula of Utricle X550. Waltzing Guinea Pig. Normal macula. Note otoliths, normal neuro-sensory epithelium and normal nerve supply.



1. Macula.
2. Otolithic membrane.

Fig. 4. Saccule X95. Waltzing Guinea Pig. Normal structures throughout. All macular structures are normal. The organ of Corti in this animal showed marked degeneration.

the sense of touch and sight. The vestibular apparatus played no part in the righting reflexes. Comparing blind normal guinea pigs with blind waltzing guinea pigs, he found that the blind normal guinea pig's righting reflexes were practically normal while the waltzing guinea pig had great difficulty in righting except when the animal's sense of touch came into play. On rotating, the normal guinea pig gave normal body position responses to the vestibular stimulation, but the waltzing guinea pig gave no response by changes of body position during or after rotation. He concluded that the waltzing guinea pig showed a complete absence of vestibular control over movements and positions of the eyes, movements and positions of the head, and over the righting reflexes of the animal.

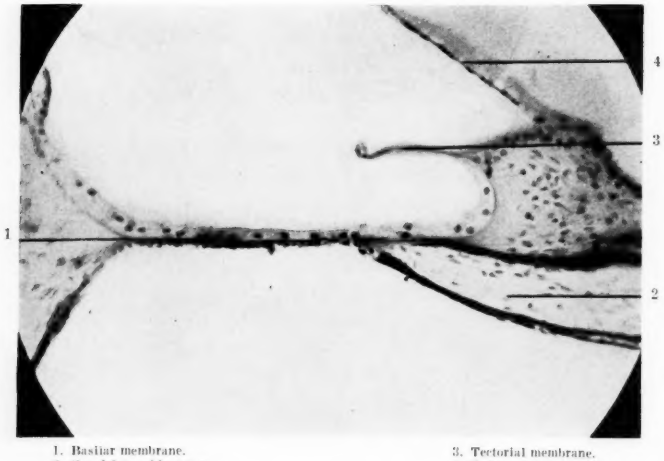
Histological examination of the vestibular apparatus of the waltzing guinea pig showed normal structures of the vestibular nerve and vestibular nuclei in all animals examined. There was no degeneration of the saccule, and no evidence was found by testing that the saccule was concerned with hearing. In none of the animals did the saccule show any degeneration of the macula even though the cochlea showed marked changes of the organ of Corti. There was also no evidence of collapse of the vestibular apparatus. A number of animals did show partial collapse but no more than in normal animals, and it was probably secondary to fixation (Figs. 1, 2, 3, 4, 5).

The evidence of a normal vestibular sense organ and normal vestibular nuclei thus pointed to the fact that the circling was probably due to a central nervous break in the reflex arc between the vestibular centers, the postural center and ocular motor eye nucleus of the animals. In experiments done to confirm this hypothesis by Lurie and Dempsey[8] it was found that injury to the interpeduncular nucleus of the brain stem resulted in circling of the normal animal in a direction opposite to the side on which the lesion was made. In their report of these experimentally induced circling animals they came to the conclusion that the destruction of the interpeduncular nucleus caused a break in the postural and ocular motor nuclei. The circling of these experimented animals was similar to the waltzing guinea pig movements, including also the position and trembling of the head. This circular motion still persisted in the experimented animals five to eight weeks after the lesion was made; at this time the animals were killed for histological study.

A. R. Buchanan[1] reporting experiments on the medulla caudal to the interpeduncular nuclei came to a similar conclusion. Lesions of the medial vestibular nucleus, medial reticular formation and the

1. Hairs.
2. Nerve to macula.
3. Neuro-epithelium.
4. Otoliths.

Fig. 5. Macula of saccule X550. Waltzing Guinea Pig. Normal structures present. Note hairs of neuro-epithelial in contact with otolithic membrane. Normal nerve supply.



1. Basilar membrane.
2. Canal for cochlear nerve.
3. Tectorial membrane.
4. Reissner's membrane.

Fig. 6. Cochlea X300. Waltzing Guinea Pig. First turn. Complete absence of the organ of Corti. Basilar membrane, tectorial membrane, Reissner's membrane are normal. Cuboidal epithelium has replaced the organ of Corti. Nerve to organ of Corti has degenerated.

medial longitudinal fasciculus resulted in circling to the opposite side. He concluded that the lesions caused a break in the connections between the postural centers and the ocular motor nuclei. In his animals the circling lasted about five to nine days, and then the animal compensated. Further experimentation will be necessary before more definite conclusions can be made.

Thus the evidence at the present time shows that in the waltzing guinea pig as in the waltzing (dancing) mouse, the vestibular end organs do not play a part in the circling. The circling is of central nervous origin probably situated in the region caudal to the mammary bodies of the midbrain.

The drum and middle ear structures are normal in these animals. Middle ear infection has been found but no oftener than in normal guinea pigs. Thus middle ear lesions can be definitely ruled out as the cause of circling or the deafness of the animal.

The deafness of the animals is inherited and is extremely interesting, for the study of the waltzing guinea pig cochlea has given clues as to the probable causes of "nerve deafness" and the way it occurs. The animals examined were not only adults but animals three to ten days old and several intrauterine guinea pigs. The adult animals were tested for the cochlear response by the methods originally described by Wever and Bray. The tests showed that the animals gave no response to sounds except for low tones up to 500 cycles per second at high intensities of 100 — 120 db. The intrauterine animals were not tested. A few of the animals ten days old were tested and gave results similar to those of the more mature guinea pigs.

Histological examination of the membranous cochlea showed that the basilar membrane, Reissner's membrane and the tectorial membrane were normal in all the animals examined. There was no collapse of the scala media in most of the animals examined. Those that did have collapsed scala media were the result of rupture of Reissner's membrane and probably caused by faulty fixation. A similar number of collapsed scala media were found in normal animals examined and were in all probability due to the same causes. We have attached no special significance to these findings as they occurred only in a very small number of animals studied. Precipitations were also found in the scala vestibuli and scala tympani but no interpretation has been made of this finding, which also occurs in the normal animals.

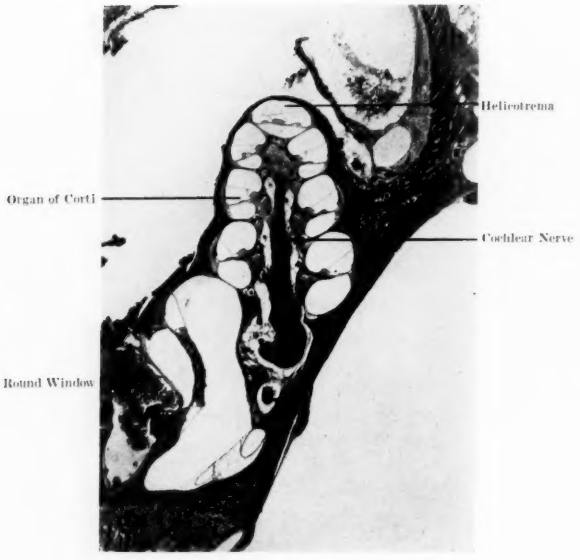


Fig. 7. Cochlea X20. Waltzing Guinea Pig. Section through modiolus. The cochlear nerve shows little degeneration present. The degeneration of the organ of Corti is most marked in the 1st and 3rd turns. There is a clot of blood in the middle ear cavity at the round window.

The stria vascularis, organ of Corti, and the cochlear nerve showed changes which we believe will give a better understanding of deafness and its causes.

The stria vascularis lies in the scala media on the spiral ligament of the cochlea. This structure is similar to the choroid plexus of the brain, and its supposed function is to secrete the endolymph not only for the scala media of the cochlea but also for the vestibular end organs. It is a network of blood vessels and epithelial cells with pigmentation. The structure, it must be remembered, runs all along the spiral ligament and is in reality very large.

In the waltzing guinea pig the stria vascularis shows all degrees of degeneration, from almost normal appearance in some parts of the cochlea to complete degeneration in other parts. The fact that there was no collapse of the scala media, saccule, utricle and semicircular canals in these animals shows that in all probability a small portion of the stria vascularis is capable of secreting sufficient endolymph to

keep the canals at their proper tension. These changes in the stria did not seem to be correlated with the changes in the organ of Corti. Degenerated stria vascularis were found in regions with normal or nearly normal organ of Corti and in other portions almost normal stria vascularis with extreme changes of the organ of Corti.

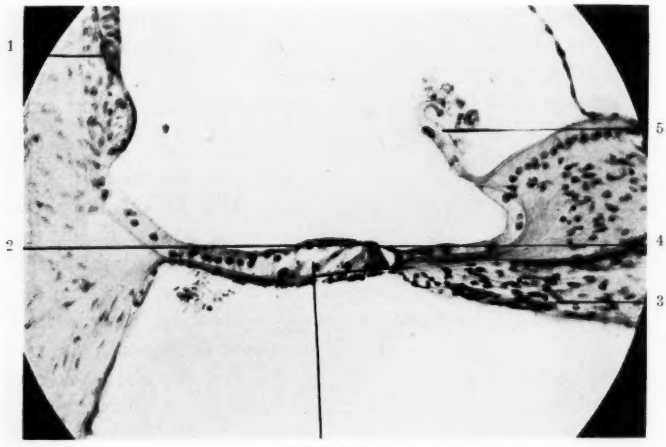
There may be in the stria vascularis degeneration of all the structures, the epithelial cells and blood vessels, and a disappearance of the pigmentation. The degeneration may involve the epithelial cells only, and the blood vessels may appear normal or be even engorged. The degrees of degeneration vary from turn to turn of the cochlea. Some of these changes have been noticed also in normal guinea pigs but not to the marked extent that it occurs in the waltzing guinea pigs. This degeneration of the stria vascularis also occurs in the new-born waltzing guinea pig and seems to occur with the organ of Corti changes.

The organ of Corti of these animals shows interesting changes not only from the viewpoints of the geneticists but also from the viewpoint of the otologist. For here is evidence of the way nature causes deafness of the inherited type and also the way the organ of Corti may degenerate as a result of disease or toxic poisons.

The degeneration of the organ of Corti may begin during intrauterine life, just before birth or in the first few days of life. The unborn animals resulting from the mating of waltzing guinea pigs were examined about ten days before delivery was expected. These animals showed normal organs of Corti and normal structures throughout the cochlea and vestibular sense organs. Two of these animals showed some possible changes of the stria vascularis and the organ of Corti in isolated turns. These isolated changes of the stria vascularis are of interest as they may be the indication of the future degeneration of the organ of Corti, though we have found no correlation of these degenerations with that of the organ of Corti changes.

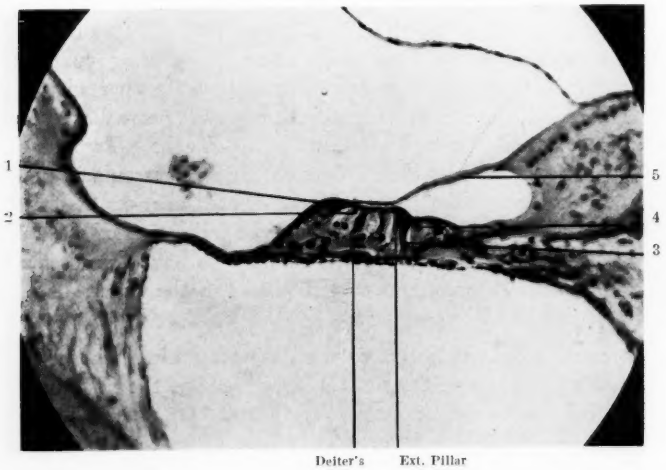
In animals examined five to ten days after birth, the organ of Corti had begun to degenerate at the apical half of the first turn and the basal half of the second turn (Figs. 1 and 7).

From this portion of the basilar membrane the degeneration spreads chiefly toward the helicotrema, not gradually but skipping parts of the turns so that portions of the third turn are nearly normal while other parts have an almost complete degeneration present. The organ of Corti in these animals appeared normal at the helicotrema and in the region of the round window. In older animals,



1. Stria vascularis.
2. External pillar cell.
3. Cochlear nerve.
4. Internal pillar cell.
5. Tectorial membrane.

Fig. 8. Organ of Corti X300. Waltzing Guinea Pig. Middle of 2nd turn. Marked absorption of the organ of Corti. External and internal hair cells have completely disappeared. The pillar cells show marked absorption and disappearance of the Tunnel of Corti. Deiter's cells also have been absorbed. Hensen and Claudius cells are still present.



1. Hensen cells.
2. External hair cells.
3. Internal pillar cell.
4. Internal hair cell.
5. Tectorial membrane.

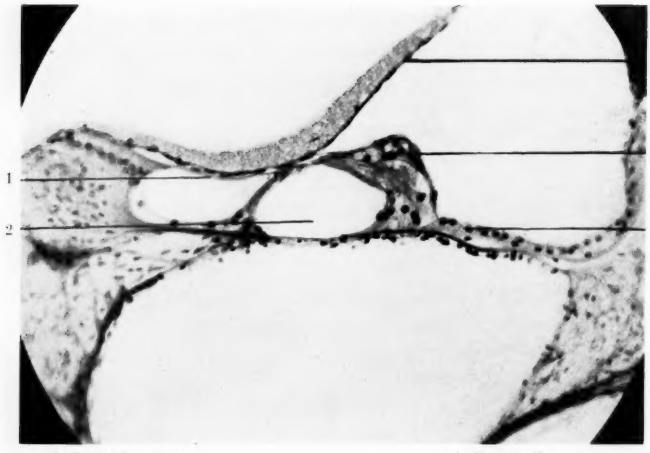
Fig. 9. Organ of Corti X300. Waltzing Guinea Pig. Third turn. Second half (helicotrema). Shrinking organ of Corti. Note internal hair cell nucleus is still present. The internal pillar cell can still be made out. The external hair cells are being absorbed. Deiter's cells are also disappearing. Hensen cells still present. Tectorial membrane normal.

where the degeneration had been going on longer, the organ of Corti in the region of the round window also degenerated, but there still remained a small portion of normal or almost normal organ of Corti at the helicotrema (Fig. 7).

This type of degeneration in an indirect manner confirms the observation of McCrady[10] on the opossum, reported in 1935. This was a report on the response of the new-born opossum to sound and its relationship to development of the normal organ of Corti. In the report he stated that hearing began 50 days after the birth of the opossum. The opossum is born as an embryonic animal and completes its development in the mother's pouch. At this time 50 days after birth the only part present of the organ of Corti of the adult type was in the apical half of the first turn. The development of the normal organ of Corti then progresses toward the helicotrema and the round window. In the waltzing guinea pig the reverse of this process seems to occur. It is of extreme interest that these observations seem not only to supplement each other but also to confirm the way the organ of Corti develops and degenerates.

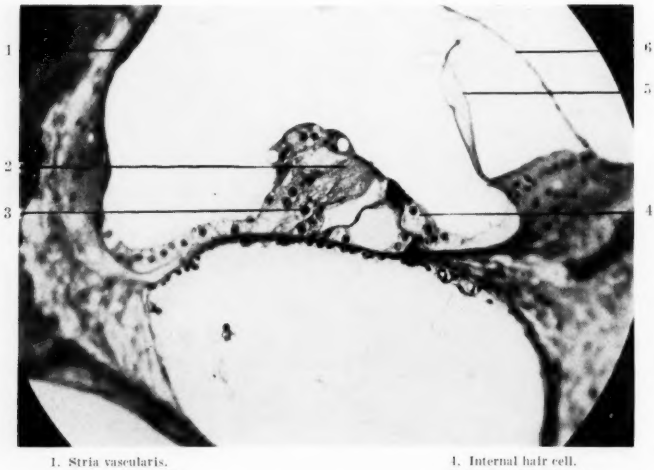
The degeneration of the hair cells and supporting cells of the organ of Corti occurs in a definite manner. The external hair cells degenerate first and are then followed by the internal hair cells. In all the animals examined we have not found the internal hair cells degenerated and external hair cells present even in a degenerated form, but have always found external hair cells absent with degenerating or almost normal internal hair cells present. The external and internal pillar cells of the tunnel of Corti seem to follow a similar order, the external pillar cells going before the internal ones. The last cells to disappear are the supporting cells. Of these the Deiters' cells go first and are then followed by the cells of Hensen and Claudius. The internal sulci cells also change gradually to the cuboidal epithelium that finally replaces the organ of Corti. There is in some portions of the cochlea a general shrinking of the organ of Corti as if the absorption was occurring all at once. When this occurs the order of cell disappearance seems to be the same, the organ of Corti being finally replaced by a cuboidal epithelium (Figs. 6, 8, 9, 10, 11, 12).

No evidence can be found as to how the absorption of the organ of Corti is brought about. Several animals were injected with trypan blue to demonstrate, if possible, the presence of endothelial cells around the organ of Corti; but results were negative, possibly due either to the filtering action of the stria vascularis or to the degeneration of the stria vascularis.



1. Tectorial membrane.
2. Tunnel.
3. Claudius cells.
4. Hensen cells.
5. Reissner's membrane.

Fig. 10. Organ of Corti X300. Waltzing Guinea Pig. Third turn. Complete degeneration of internal hair cell, external hair cells and pillar cells. The tunnel is still present and nerve fibres are still going through the tunnel. Hensen and Claudius cells are still present.



1. Stria vascularis.
2. External hair cells.
3. Deiter's cells.
4. Internal hair cell.
5. Tectorial membrane.
6. Reissner's membrane.

Fig. 11. Organ of Corti X300. Waltzing Guinea Pig. Third turn of cochlea. External hair cells have degenerated. Beginning degeneration of Deiter's cells. Internal hair cells and pillar cells appear normal. Note that organ of Corti still retains its normal general outline and no general absorption has taken place. Stria vascularis has atrophied.

It is important to note that this definite manner of degeneration of the hair cells of the organ of Corti has also been found in animals made deaf by exposure to loud tones and by toxic poisoning. Here, also, the external hair cells degenerated first and then the internal hair cells. This confirms observations made and reported previously as to a difference in the development and function of the internal hair and external hair cells.[5, 6, 9]

The spiral ganglia and the cochlear nerve also degenerate but at a much slower rate of speed. In a great many of the young animals there were still nerve fibres in almost normal abundance coming from the basilar membrane, though there was no organ of Corti present. The spiral ganglia also appeared normal. Later the nerve and ganglia degenerate and there are finally left in the cochlea a few scattered ganglia cells and a small amount of nerve fibres in the 1st, 2nd and 3rd turns. At the helicotrema (4th turn) there may be present a partially degenerated or almost normal amount of ganglia cells and nerve fibres. There are as a rule more nerve fibres and ganglia cells present throughout the cochlea than one would expect from the amount of organ of Corti present. The cochlear nerve is smaller than normal as it enters the medulla, but no degeneration or abnormality of the cochlear nuclei was observed even when special nerve stains were used (Figs. 13 and 14).

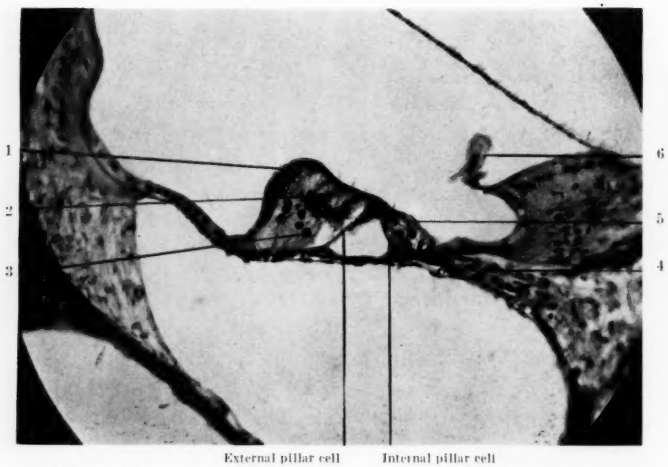
Throughout all these changes the tectorial membrane remains normal, and no changes of any kind could be found in its structure and position. All other structures of the cochlea were found to be normal. There were no bone changes noticed of the cochlear capsule or ossicles. The lesions found were limited to the stria vascularis, organ of Corti and its nerve supply.

CONCLUSION

1. These animals inherit deafness and waltzing (circling). The inheritance follows the mendelian laws for recessive characteristics.

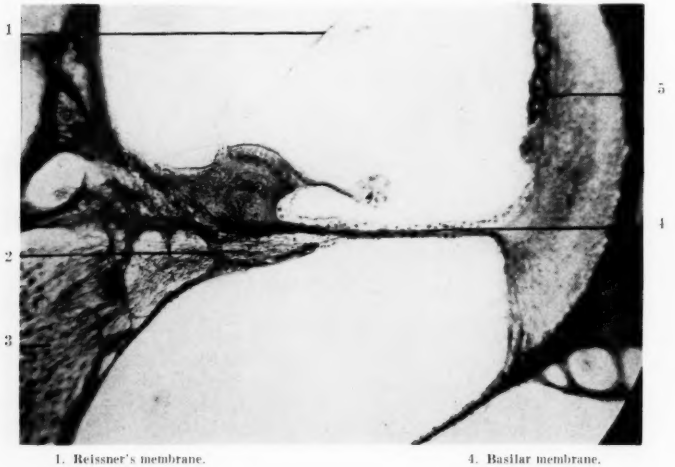
2. The vestibular sense organs, the utricle, semicircular canals and saccule do not cause the waltzing (circling) of the animal.

3. The waltzing (circling) is caused by a central nerve lesion which can not be demonstrated by histological study. All proof obtained at present for the cause of this waltzing (circling) has been by animal experimentation. The interpeduncular nuclei are believed to be the possible site of this waltzing (circling).



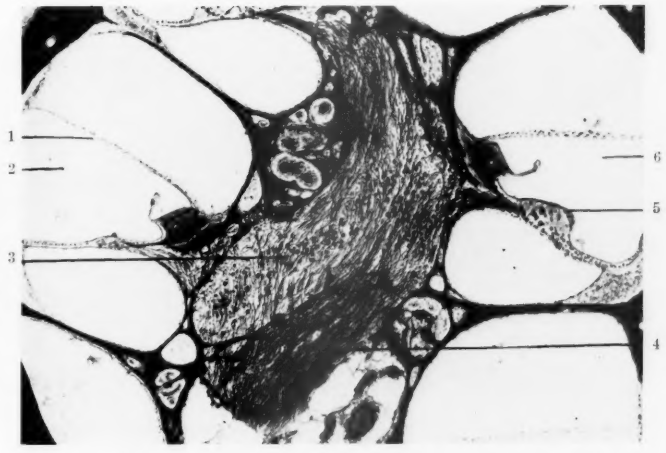
1. Hensen cells.
2. External hair cells.
3. Deiter's cells.
4. Cochlear nerve.
5. Internal hair cell.
6. Tectorial membrane.

Fig. 12. Organ of Corti X300. Waltzing Guinea Pig. Fourth turn at the helicotrema. The organ of Corti shows some degenerative changes but appears practically normal. Note the hairs of the hair cells still present and the normal nerve supply.



1. Reissner's membrane.
2. Nerve fibres.
3. Spiral ganglion.
4. Basilar membrane.
5. Stria vascularis.

Fig. 13. Cochlea X160. Waltzing Guinea Pig. First turn. Bodian silver stain to show nerve fibres. Organ of Corti completely absorbed and replaced with cuboidal epithelium. Nerve fibres still present going to spiral ganglion. Animal 10 days old.



1. Reissner's membrane.
2. 3rd turn.
3. Spiral ganglia.
4. Cochlear nerve.
5. Organ of Corti.
6. 4th turn.

Fig. 14. Cochlea X100. Waltzing Guinea Pig. Third and fourth turns. Bodian stain to show normal nerve fibres. The organ of Corti is not stained well by this method. Note degeneration of organ of Corti of the third turn. Nerve fibres are still present going to this degenerated organ of Corti. The organ of Corti of the fourth turn shows slight degeneration, but the nerve supply is normal in amount and appearance. The ganglia cells are normal.

4. The deafness of the animals is caused by degeneration of the organ of Corti.

5. The degeneration of the organ of Corti occurs in a definite manner, the external hair cells degenerating first and the internal hair cells later.

6. The cochlear nerve and spiral ganglia cells degenerate at a much later period.

7. The stria vascularis shows definite degenerative changes, but they have not been correlated with the changes found in the organ of Corti.

8. The macula of the saccule did not give evidence of being concerned with hearing, either by degenerating with the organ of Corti or by responding to sound stimulation.

483 BEACON STREET.

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XI

PROGRESS IN THE CONSERVATION OF HEARING

HORACE NEWHART, M.D.

MINNEAPOLIS

The purpose of this paper is to present facts pertaining to the conservation of hearing which should have greater publicity in order to accelerate activity in the solution of the problem.

The deaf, that is, those in whom the sense of hearing is nonfunctional for the ordinary purposes of life, are well provided for in all of our states by wise legislation. A vastly larger group, the hard of hearing, have received relatively scant attention.

Accomplishment in the prevention, treatment and amelioration of hearing impairment as compared to achievement in other medical fields, such as ophthalmology, has been slow. Appreciation of normal hearing for the educational, economic and social requirements of life has been increased by the advent of the audiometer. Its application in research and diagnosis has revealed new possibilities of attainment. Nevertheless effective, organized effort in solving the problem has been limited, sporadic and until recently, discouraging.

The situation merits careful, unprejudiced scrutiny and a frank appraisal of the status of the problem, the solution of which has been retarded by many obstacles. The chief hindrance to greater attainment has been a profound lack of knowledge of the fundamental facts pertaining to the subject. A wider dissemination of this knowledge among medical practitioners, educators, public health and school nurses and parents is urgently needed.

Informed groups in various communities have successfully stimulated a sincere desire for greater achievement in this field. This interest constitutes a definite challenge to physicians, especially otolaryngologists and pediatricians, to accept a new responsibility and perform actively their recognized function as leaders and teachers of the public in health education as related to safeguarding the hearing.

Among the many obstacles to progress in this field of preventive medicine we mention the following facts:

1. It is not generally known by the laity that a person may lose a very considerable amount of his original hearing without being aware of the loss. The consequent delay in seeking medical aid and the frequent failure to obtain satisfactory results has caused the widespread misconception that treatment for ear conditions producing hearing impairment is largely futile. The persistence of this idea, in spite of the fact that it is now recognized that a large per cent of cases of handicapping hearing loss are preventable or capable of arrest is largely responsible for the indifferent attitude still prevailing in many communities toward organized effort to prevent hearing impairment.

2. Extensive application of the audiometer has disclosed an unexpectedly high incidence of significant hearing deficiencies among our population. By a significant hearing deficiency is meant any hearing impairment capable of interfering with the acquisition of normal, articulate speech, a fair education and a personality which will ensure to an otherwise normal individual his economic and social security.

3. The use of the audiometer has proved:

a) That the older means for the detection and measurement of hearing defects, the watch tick, acoumeter, whisper and conversation voice and tuning forks as usually applied are too crude, inaccurate and time-consuming to meet the exacting requirements of modern otologic practice;

b) That tests to detect slight but significant hearing impairment are not accurate if made in the presence of interfering noises; and

c) That to determine the degree of a severe defect in one ear the better ear must be masked. These technical details are mentioned because they must be impressed repeatedly on nurses and technicians who apply the tests.

4. Educators and parents should be definitely informed that among young school children hearing deficiencies so slight that they are unnoticed by teachers and parents often cause retardation, defects of speech and social and emotional maladjustments. Such defects, unless discovered early and corrected, can ruin many a child's career.

5. Recently Drs. Crowe and Guild and their associates have shown that losses for the higher tones beyond ordinary speech frequencies are caused by hyperplastic lymphoid tissue about and within the eustachian tubes. If within the tubes, the tissue is inaccessible for surgery but curable by radon irradiation. If discovered and treated early, improvement in the hearing often results.

This contribution widens our conception of the pathology of hearing impairment and calls for a revision of our technic for the discovery, prevention and treatment of hearing deficiencies in school children. It suggests the advantage of using the pure tone audiometer for screening tests. It further explains certain cases of persistent progressive hearing impairment after tonsillectomy and repeated adenoidectomies, and adds confirmation to the teaching that many of the handicapping hearing defects which first appear gradually in adult life result from forgotten nasopharyngeal, tubal and middle ear infections early in life.

6. The public is sadly uninformed as to many possible causes of hearing impairment due to pathology or dysfunction involving parts of the body remote from the ear, and to external causes. The remedy is to be found in more and better health education regarding the care of the ears, beginning during school life.

The too common practice of consoling an older person by telling him that his increasing hearing handicap is due to his years is to be deprecated, since the diminution which can be justly attributed to a physiological loss is slight, involves the higher frequencies and is not handicapping for the perception of person-to-person conversation. Other causes have been operative.

7. An insufficiently recognized hindrance to a better understanding of the problem of preventing and ameliorating hearing defects is the almost universal but illogical and confusing use of the terms "deaf" and "deafness" tolerated by long, indiscriminate usage. In the opinion of an increasing number of otologists and educators of the deaf the word "deaf" used as a collective noun should be limited to indicate only "those in whom the sense of hearing is nonfunctional for the ordinary purposes of life."* The adjective "deaf"

*This definition adopted by the 1937 Conference of Executives of Schools for the Deaf was approved by resolutions adopted by the American Otological Society of the American Academy of Ophthalmology and Otolaryngology at their last annual meetings. It should be seriously considered by physicians, educators, editors and lexicographers for incorporation in the terminology of otology.

and the noun "deafness", therefore, consistently should be used only to indicate the condition of those who are thus seriously afflicted.

Those in whom the sense of hearing, although defective, functions with or without a hearing aid, should be designated as hard of hearing, deficient hearing or acoustically handicapped.

We do not call those with visual handicaps correctible by glasses "blind" nor their condition "blindness." Why discriminate against persons with hearing deficiencies which can be compensated by hearing aids by calling them "deaf" and their deficiency "deafness" since both terms imply a justly resented stigma? This is a frequent cause of the persistent effort to conceal their infirmity and to postpone resort to medical and mechanical help.

An ideal, practical plan for conserving the hearing, adequately covering the entire field on a state-wide basis, has not been put in successful operation. The administration of such a program will involve legislation providing for readjustments of the activities of various educational, public health, welfare and social agencies and for increased appropriations for which the people must first be prepared. We cannot reasonably expect immediate radical action to correct completely the present backward situation while interest is so intently centered on national preparedness.

This should not preclude every reasonable effort to advance the cause of better hearing at this time. Measures to conserve the hearing should be included with other health measures essential to a comprehensive preparedness program, recognizing the fact that increased hazards to hearing exist in industry, aviation, naval and military operations in time of stress and that the man with defective hearing is a dangerous liability.

The public in many progressive communities has already become ear conscious and sees the larger possibilities for initiating measures which will promptly yield worthwhile returns at a minimum cost. The immediate need is initiative and leadership which must come from the medical profession, understanding educators and welfare groups. This has been the case in all successful health movements.

New York State has been outstanding in its accomplishments in this field. Within the last five years it has enacted legislation requiring (1) physicians, nurses, parents and guardians to report to the State Commissioner of Health every child under six years of age who is totally deaf or whose hearing is impaired; (2) the annual testing of the ears of all school children by means of audiometers approved

by the Commissioner of Education; (3) the appointment of a competent person to supervise audiometer tests; and (4) instruction in normal schools in the best methods of making hearing tests.

In 1936 this state created an able temporary commission "to examine, report upon and recommend measures to improve facilities for care of hard of hearing and deaf children and children liable to become deaf". The three published reports of the commission are highly informative. The above activities were the result of the persistent efforts of otologists, the organized hard of hearing and interested friends. Creditable beginnings have been made in several other states and in many widely distributed cities and smaller communities.

It is now accepted that of all procedures for effectively conserving the hearing of our population, the first and most important one is the incorporation in every school health program of the periodic testing of the hearing of all pupils by the audiometer. This makes possible the early discovery of those who have threatened or handicapping hearing defects that they may have the prompt application of needed medical care and educational readjustments.

Warren H. Gardner, Ph.D., Chairman of the Committees on the Hard of Hearing of the American Society for the Hard of Hearing, in his report for the school year 1938-39 states that 1,871,000 children were given audiometer tests in 767 cities and towns and 125 counties. This is a remarkable gain over previous years. The number in New York State was 590,000. Massachusetts gave 180,000 tests in 171 towns and one county, California, 155 tests in 42 towns and 9 counties. In Pennsylvania, where the State Department of Education two years ago purchased 47 audiometers to be loaned to school units which did not own the equipment, 107,000 were tested last year. Indiana and Oklahoma each tested 70,000. W. P. A. surveys in Ohio and one county in Illinois accounted for 140,000. This is a fine beginning, but in many states and larger cities the procedure is practically unknown. The incidence of a significant hearing loss was between six and seven per cent with wide extremes dependent on many variable factors.

The cost of special equipment and personnel has been the most serious obstacle to the general adoption of this procedure. Other causes of delay have been unfamiliarity with the purpose and technic of the tests and their practical value.

The high price of audiometers, prohibitive for many schools, lately has been materially reduced. Co-operative ownership among

several neighboring communities has resulted in a substantial saving. The use of recently available, low cost, reliable, pure tone school audiometers applicable both for the screening and the final tests of pupils of all ages has reduced the cost in communities in which the school population is under 3,000. In many places the cost for operating personnel is kept down by using experienced volunteer helpers or those provided by local social service groups as the Kiwanis, Rotary and similar organizations whose members in some instances also provide equipment.

The advantages of this procedure to the individual through the preservation of his hearing and to the public in a reduction of taxes for special education, vocational training, rehabilitation and relief of acoustically handicapped unemployables more than compensate for the cost.

Inconsistent discrepancies in the incidence of hearing deficiencies occasionally reported by different communities have caused some doubt as to the value of the tests. These differences are explained by failure to follow implicitly the operating instructions of the manufacturers and to employ uniform criteria for referral of deficient hearing pupils for otolaryngological examination. To insure greater accuracy it is recommended that for the present the practice adopted by the New York State Education Department be followed.

The phonograph audiometer, almost exclusively used for group screening tests, has certain limitations with which every physician and school executive should be familiar.

1. It does not reveal hearing deficiencies for the higher frequencies which are now considered important as an early indication of tubal pathology.

2. It cannot be used satisfactorily in testing children too young to write dictated numbers. As a result there has been a trend toward the use of pure tone or pitch-range audiometers for testing individually pupils of all ages.

We do not recommend abandonment of the phonograph audiometer in schools in which it is in use, thereby discrediting a valuable instrument which has served and will serve a most useful purpose. We advise its continued use for testing older pupils when large numbers are involved, supplementing it by a pure tone instrument for the final individual retest as recommended by otologists experienced in this work.

Deserved criticism is made of the inadequacy of the important medical follow-up of pupils found to have hearing deficiencies. This failure is due to insufficient funds and lack of co-operation between the medical profession and local welfare agencies. The school's responsibility should be limited to the discovery of hearing-defective pupils, provision for their special educational needs and persistent effort to secure for them competent medical care. In rural areas without effective school health programs this is difficult. There is special need for Federal aid which is available under certain conditions.

Before a comprehensive plan for conserving the hearing can be put into successful operation, greater initiative and leadership by otologists and interested, civic minded citizens is needed to carry on an organized educational program which will result in effective legislation.

A significant step, far reaching in its possibilities, has been taken recently by the American Society for the Hard of Hearing in the creation of a Committee on Legislation for the Hard of Hearing. The chairman is Mr. Conrad G. Selvig, an able educator and a former Congressman from Minnesota, now residing in Santa Monica, California. The organization of carefully selected state committees already has been effected. Minimum state legislation as outlined by Mr. Selvig covers:

1. Compulsory hearing tests for all children at regular intervals by competent scientific methods.

2. Proper medical follow-up treatment and care for all in need, and instructions to parents as to its necessity.

3. Remedial instruction in the form of lip reading from primary grade through college. Special training in speech and voice where necessary. Group hearing aids in every school where there are five or more hard of hearing children.

4. Vocational guidance.

5. Adult classes in lip reading serving the hard of hearing in all communities in the state.

6. Assistance to the hard of hearing in search of employment and a fair deal under Civil Service rules.

7. A full-time director of work for the hard of hearing on the staff of the State Department of Education.

8. Training courses for teachers of the hard of hearing in every State Teachers' College.

We would add one more: Safeguards to protect workers in industry from the effects of loud noises capable of causing damage to the hearing.

A better understanding of the problem of conserving the hearing will be attained by providing in extension and refresher courses for practicing physicians and public health workers information covering the newer developments in otology concerning the discovery, prevention and amelioration of hearing defects as related to the school health program and to private practice.

Not all of the objectives for which legislation is proposed can be achieved in every state at once. The minimum initial objective to be demanded in all states is provision for regular audiometer tests of all school children to be administered by the Department of Education in cooperation with the State Board of Health. To insure uniformity and effectiveness in this work adequate funds should be provided for a qualified supervisor of hearing tests and for the purchase of audiometers to be loaned on a low rental basis to communities not able to purchase their own.

CONCLUSION

Substantial gains already have been made in effective work in this neglected field of preventive medicine. More rapid achievement now depends on a thorough knowledge of the problem and increased interest and leadership from an understanding medical profession.

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XII

TINNITUS AURIUM IN THE LIGHT OF RECENT RESEARCH

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Recent audiometric studies of tinnitus suggest many interesting questions as to the physiological and patho-physiological implications of this symptom of disease. Certain speculations and interpretations are justified in the light of the facts and current theories. Only after the advent of a satisfactory audiometer in 1922 has it been practical to obtain dependable quantitative data as to the intensity, frequency, and masking characteristics of tinnitus. This discussion is based on routine measurements made since that date. I shall discuss mainly some of my own observations and interpretations.

1. There are two fundamental types of tinnitus, vibratory and non-vibratory. Vibratory tinnitus is mechanical in origin. It is caused by an actual to-and-fro response of the media in the ear, to vibrations. It may arise from vibrations without or within the ear or be brought to the ear through the solid, liquid, or gaseous media of the body. Common causes are muscle spasms, vascular bruits from the venous or arterial channels and nasopharyngeal squeaks, clickings, droppings, etc.

Non-vibratory tinnitus is not mechanical in origin. It is caused by a biochemical (non-vibratory) irritation of the auditory neural elements. It is an illusion of sound. The impulses set up are similar to the impulses set up in other nerves by changes in environment. Such changes may occur directly through pressure, degenerative or regenerative, anoxic, toxic or traumatic lesions, and indirectly by disorders of the endocrine glands and of the autonomic nervous system. Vibratory and non-vibratory tinnitus may occur separately or together. Herein it is with the non-vibratory tinnitus that we are particularly concerned.

2. The loudness of tinnitus may be estimated objectively by comparing it with an applied tone or noise of similar but known loudness, that is by balancing the tinnitus with the sound being used for comparison. This is called determining the "loudness balance". It

was in part my attempts to measure tinnitus that led to the discovery that in some ears there is present more than the expected progressive increase in loudness, as the intensity of a sound is increased. In other words, as compared with an opposite normal or obstructively deaf ear, I discovered that a 20-decibel increase in the sound in a nerve-deafened ear appears like a greater increase than 20 decibels. It often appears more than a 40-decibel increase and even more than a 60-decibel increase. I also noticed that the rate of increase in loudness accelerates rapidly with intensity so that for very loud sounds there is often little or no difference in the sensation of the loudness when the same loud sound is applied to one, a normal, or the other, a nerve-deafened, ear. Although more accurate estimates of loudness may be obtained by comparing similar frequencies it is not absolutely necessary to use similar tones. Any two sounds may be compared as to their relative loudness.

The increase in the increment of loudness with intensity I have called the "recruitment phenomenon". It has proven to be the most positive and reliable sign of so-called nerve deafness. It is absent only in the presence of pure conduction deafness or normal hearing. It, of course, cannot be elicited by the binaural loudness technique if one ear is totally deaf, and it is therefore evident that its absence when one ear appears extremely deaf is a definite sign of total deafness in such an ear. The "recruitment phenomenon" may be demonstrated monaurally by using the loudness balance technique at two frequencies showing a difference in hearing loss of at least 10 decibels. It may be elicited binaurally by the alternate or simultaneous applications of measured sounds to the better and the worse ears. In an ear in which there is a difference in the degree of nerve deafness in any portion of the frequency scale the "recruitment" will be greatest at the more nerve-deafened frequency area.

Tinnitus is usually uncontrollable except by masking. It is impossible to turn it on and off at will. For greater accuracy this makes it advisable and usually necessary to measure tinnitus in comparison to a sound heard in the opposite ear, i.e., balancing it in loudness with a tone applied to the opposite ear.

3. It was frequently found that although a patient complained of intense tinnitus, in many instances measurements might reveal that it required less than 10 decibels above threshold to equal the tinnitus in loudness (often less than five). Studies are now being made to determine the reason for this seeming paradox.

4. The frequency band or tone of a tinnitus may be objectively determined by a comparison with similar applied frequencies. The

measuring sound should be intermittently applied, and preferably to an opposite normal or obstructive deafness ear. A too near coincidence in frequency may, by summation, make the tinnitus appear louder. In other words the two sounds (tinnitus and applied sound) will appear as one, or if the measuring sound is louder than the tinnitus only the measuring sound may be heard. In either case the tinnitus and the measuring sound cannot then (beats being absent) be satisfactorily compared to measure the frequency of the tinnitus.

Binaural tinnitus equal in intensity and frequency, as might be expected, appears to increase the sensation of its loudness. Applied frequencies simultaneously and equally heard by the two ears will do this. With these reservations the nearer the applied sound approaches the tinnitus in loudness and frequency the more accurate will be the measurements. In difficult instances, begin by applying discrete frequencies alternately, well above and well below the frequency tone or band of the tinnitus. Then gradually narrow the spread between the two exploring tones until they meld into or narrowly bracket the frequency band of the tinnitus.

5. Tinnitus may be measured in relation to its maskability by discrete or by band frequencies (thermal noise).* It is a common occurrence to have a patient say that his tinnitus is not heard in the street, a bus or railway trains, etc.; also to have others say that they never lose their head noises—that even in the noisiest surroundings they still hear them. This illustrates roughly the difference in masking effects of environmental sound in different ears.

The ease of masking of tonal tinnitus often varies greatly with the different frequencies used for masking it. Chart I shows two instances wherein the masking was easily accomplished at all frequencies. This is the more common experience even though the frequency of the tinnitus may appear to be limited to a narrow band on the frequency scale. It may appear to the patient to be a definite tone, as though made by a tuning fork or a whistle. Chart II shows an instance wherein the masking was greatest as the masking tone approached near to the frequency band of the tinnitus; also shown is the level of masking by the thermal noise.

6. Neither the loudness of tinnitus nor its frequency alone determines the ease of masking it. Chart III shows two cases wherein the masking was more difficult at all frequencies than the loudness of the tinnitus would be expected to warrant. Chart IV shows two

*The thermal noise used for masking should contain all frequencies in equal loudness. It is then a hissing sound like the sibilant "sh".

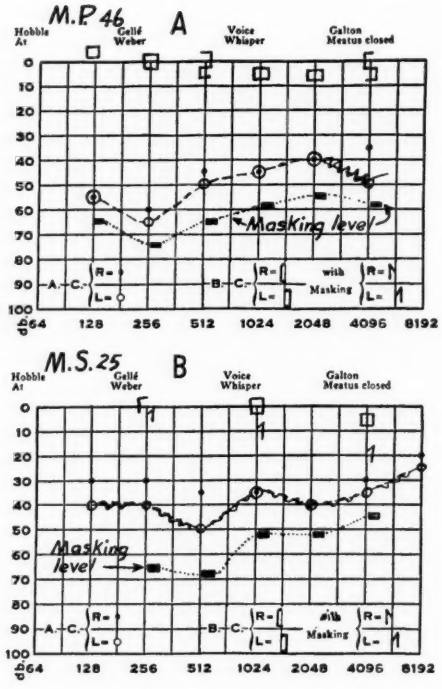

Chart I. Audiograms in two cases of otosclerosis. The patient in Case A suffered from a pulsating and roaring, escaping air tinnitus in the left ear, of 10 to 15 decibels in loudness. This tinnitus was easily masked at all frequencies, 10 to 15 decibels above the patient's threshold of hearing, and by thermal noise. I believe that both the deafness and the tinnitus were of peripheral origin. Note that bone conduction was diminished only 5 decibels. It may have been originally high normal in which case it would now appear near normal even in the presence of a 15 to 20 decibel nerve deafness.

The patient in Case B complained of a constant blurred whistling tinnitus in the region of 3000 in the left ear. This was felt as present even when not actually heard. This tinnitus was masked by 25 decibels over the threshold of hearing at 256 double vibrations and by progressively diminishing intensities at frequencies 512, 1024, 2048, and 4096. It was easily masked by thermal noise. The hearing improves when the tinnitus ceases and goes down about 10 decibels in the frequency areas of the tinnitus when it is present. I believe that the deafness and the tinnitus are of peripheral origin. Note that bone conduction is increasingly lowered in the left ear with frequency, even though the reverse is true of air conduction. This indicates for the higher frequencies a combined nerve and conduction deafness.

instances wherein the masking was impossible even when using very loud sounds.

If the loudness of tinnitus and the difficulty in masking it always ran parallel (as on first thought one would expect) there would be little difficulty in interpreting the masking experiments. In other words a parallel response suggests that as the loudness of the tinnitus corresponds to the number of nerve elements activated by the irritation of the lesions, the relative difficulty in masking the tinnitus corresponds to the number of those nerve elements beneath the basilar membrane (or elsewhere) which are still available for further stimulation.

Only those elements in the nerve areas producing the tinnitus would be expected to produce a sensation indistinguishable from the tinnitus. The lesions causing tinnitus need not necessarily correspond exactly to the tonal distribution of the tinnitus or to the frequencies most affected by the deafness, but they are usually found within such areas unless the deafness is very severe. The tone of a tinnitus depends also upon the pattern and degree of the neural degeneration.

7. It was found that the presence of tinnitus is always associated with more or less deafness. This of course was to be expected when one realizes that neural impulses, whether set up by mechanical irritations as from sound vibrations or by biochemical irritations as from pressure, trauma, PH changes, etc., always monopolize the neurons activated. These wires are busy and so cannot be used by superimposed irritations. Therefore they are, while occupied by tinnitus impulses, useless for activation by sound vibrations, and the amount of deafness the tinnitus impulses impose depends upon the number and location of the "busy wires" (neurons).

Any audible sound elevates the threshold more for all sounds near it in frequency than for sounds further removed from it in frequency. This also holds good for tinnitus. With tinnitus, as with applied frequencies, masking is usually greater near and below the frequencies being masked (Chart II). There is a large spread in the neural activations from even a fairly loud sound, and therefore there is a wide spread in the "busy wire" effect. Masking tinnitus with an applied sound in many circumstances shows a similar relationship between loudness and masking to that obtained by masking one frequency with another. The exceptions will be discussed anon.

8. The hearing, especially "speech hearing", fluctuates indirectly with changes in loudness of the tinnitus (Chart V). Hearing vari-

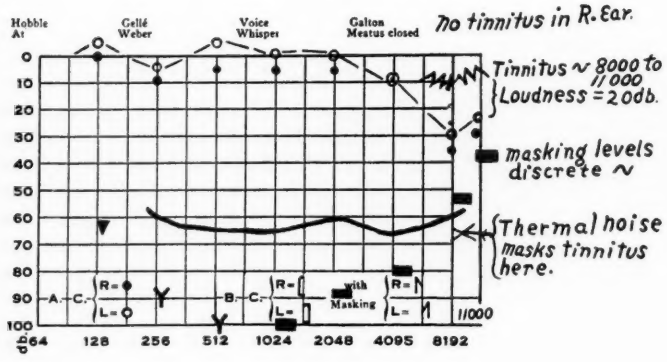

Chart II. The tinnitus in the left ear sounds like the high frequencies of thermal noise (like myriads of insects). The masking by discrete frequencies is close to the pattern obtained in masking applied sounds similar to the tinnitus. It occurred only above 512 and then progressively the closer it approached the frequency band of the tinnitus. The speech frequencies are little affected by the tinnitus, because the tinnitus frequency band is above them. The bone conduction is normal for all frequencies up to 4096, above which, in most persons, it is not dependable because it is heard less by way of the bone than by way of the air conducting mechanism. Thermal noise masked the tinnitus at 65 decibels over normal thermal noise threshold. This is 35 decibels over the threshold of the ear at the tinnitus frequencies. It may be that the high intensity required for masking is due to a lack of sufficient loudness in the thermal noise frequencies above 9000. A frequency of 8192 masked the tinnitus at a setting of 55 decibels. This is 25 decibels over the threshold at 8192 and 5 decibels greater than the loudness of the tinnitus. At 11,000 masking occurred at 15 decibels over the threshold. There is at times a faint tinnitus in the right ear, similar in tone to that in the left ear. A mixed neural origin, peripheral and central, for the tinnitus, with associated high tone deafness, is suggested. A slowly progressing deafness for the high tones has been recorded during the past 20 years. This appears to be associated with fatigue and the slowly progressing degeneration. The tinnitus varies at times from 2½ to 30 decibels in loudness and sometimes is said to appear to harbor two distinct tones, superimposed on the background of the general high hissing sound, one very loud between 500 and 1000 D.V. and one near 12,000.

ations depend also upon fluctuations of the frequency band of tinnitus. Both of these phenomena would be expected to follow from what has been set forth above.

Although the frequency and loudness of tinnitus determine the deafness caused by the irritative lesions setting up the tinnitus and by the tinnitus itself, they do not necessarily coincide with the deafness in general (Chart V). Lesions similar to those causing the tinnitus may cover wide areas wherein they produce no tinnitus. This observation is supported by the fact that even though tinnitus be limited to narrow frequency areas, when the tinnitus ceases, the hearing often improves not only in the former tinnitus areas but over wide spreads of the audiogram curve (Chart V). If the lesions causing the tinnitus progress to complete mass degeneration of the neural elements, the tinnitus, unless continued from other locations, will cease entirely. In this case the hearing loss at the former tinnitus frequences will not be recovered.

9. Fatigue (deafness) from tinnitus or the irritations causing tinnitus is important, but as yet nothing definite is known about it. Any deafness produced by fatigue from biological irritations would appear to be somewhat different from that caused by fatigue from actual vibrations. In the first place tinnitus seldom attains anywhere near the intensity which we use experimentally to produce fatigue. Tinnitus is usually faint and therefore well below the intensity required to cause acoustic shock. However, it is well known that constant biochemical activations of any nerve cause fatigue and an increasing fatigue with time and constancy. It is probable that even when biochemical irritations are too slight to produce audible tinnitus they may nevertheless produce some fatigue. Ordinarily the tinnitus fatigue factor is slow-acting and varies with the etiology of the tinnitus. I have observed several patients with almost constant tinnitus (of from 5 to 20 years' duration) who show a slow progressive loss in the hearing at the tinnitus frequencies but little or no change at the other frequencies. General neurological fatigue and hypersensitiveness caused by the tinnitus, in some people is also a very important factor because it tends to accentuate the tinnitus and the annoyance it occasions and therefore also the deafness. These observations correlate with observations in other fields of sensory perception.

10. Attention is called to the fact that on sensitive areas of the skin slow vibrations may be quite accurately differentiated through touch alone (there is no frequency place representation). It would then appear possible if not probable that the highly developed touch

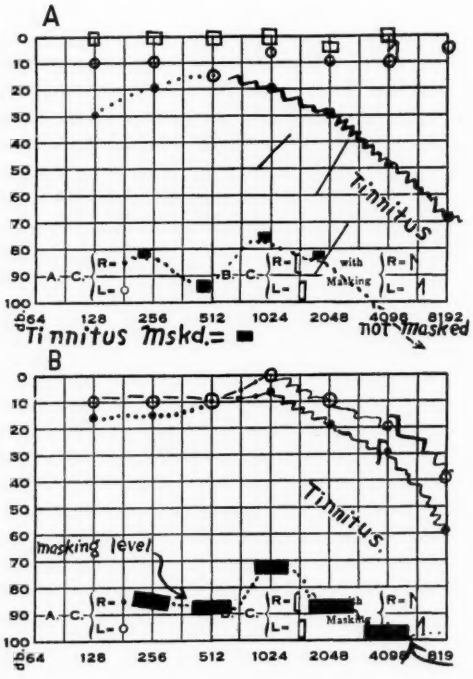

Chart III. Audiograms in two cases of healed otitis with evidence of scar tissue in the middle ear and a marked loss of hearing for the high tones in the worse ears. In both patients the tinnitus was high pitched and masked with difficulty at all frequencies; as shown in the graphs, it required from 80 to 90 decibels above the normal threshold to obliterate the sound of the tinnitus. It is my opinion that difficulty in masking is due to the fact that the tinnitus is caused not by actual vibrations in the cochlea but by some biochemical change in the neural pathways. In order to eliminate the possibility of the tinnitus being heard also in the ear not masked, binaural masking was also employed, with no change in the results. The patient in Case A has hyperthyroidism. At the tinnitus frequencies it was impossible to mask the tinnitus with the amplitudes available. Masking noise in the opposite, normal ear also did not mask the tinnitus. At times, when the masking tones are very loud, this patient's tinnitus alternately appears and disappears. It may be that this is influenced by the auditory reflex action on the intra-tympanic muscles.

The patient in Case B had a history of gastrointestinal disturbances, bilateral tinnitus, vertigo for two years and no noticeable deafness for speech. (Neither of these patients gave a history of otitis media, in spite of the evidence of scarred drums.) It may be that in both cases the difficult masking by thermal noise is due to lack of intensity of the high tones (above 9000) in the masking apparatus, making it less efficient in masking very high tones. However there is little or no tendency to increasing masking with frequency, which in my opinion, when present indicates peripheral lesions. Moreover, apparently similar ears are commonly encountered wherein masking is accomplished at all frequencies at 5 to 20 decibels above the threshold. It would appear that in the two cases illustrated the lesions causing the tinnitus, in part at least, are not peripheral. They are therefore believed to be in the medial or central neural pathways.

mechanism of the cochlea should also be able to differentiate at least the lower frequencies one from another, without absolute dependence upon the mechanism of frequency spatial areas. This would coincide with the findings of Wever and Bray and others.[1, 2] I infer from all this that whereas normally, spatial responses are involved for a full tonal quality from mechanical vibrations, abnormally, as from the irritations of lesions causing tinnitus, the firing rates of the nerves to the various areas will more or less persist in the spatial areas involved. As long as there is life the nerve will fire when irritated. Only the presence of degeneration of the nerve elements in the end organ, ganglion, neuron or collaterals, central ganglion, etc., can prevent the possibility of stimulation and firing of the elements in question. Be all this as it may, a universally accepted solution is not yet established, and it may well be that both mechanisms have a part in the sensing of tone.

11. Increases in the loudness of tinnitus are often attended by an elevation in tone and vice versa. Similarly if any band of applied frequencies is increased in intensity it will tend to sound to the ear somewhat higher in pitch. This is probably due to the increased spread of the activated neural elements in the cochlea, and as even an equal spread up and down involves stimulating proportionately more of the high tone areas than the low, the overshadowing sensation will lie more within high frequency components. This is one of the reasons why to some deafened ears even moderately loud sounds appear higher in pitch (not more definitive but higher in tone sensation) than to the opposite normal ears.

12. Beats between a tinnitus and an applied pure tone cannot be demonstrated unless the tinnitus is vibratory (extra-neural) in origin. Beats when demonstrable would therefore be proof that the tinnitus is of extra-neural origin. In my opinion spontaneous rhythmic mechanical vibrations cannot arise from the hair cells or any of the neural elements of hearing.

There is another sure sign of vibratory tinnitus, namely, its audibility to the examiner. Only actual vibrations (not biochemical activations) can be heard by someone other than the subject. Some are often easily heard, such as hemic and other heart and vessel murmurs, and mechanical noises originating in or brought to the ear from the eustachian tube and nasal and other respiratory passages.

Sound vibrations may reach the ear from any part of the body and if they are of definite frequency, and then only, may be utilized

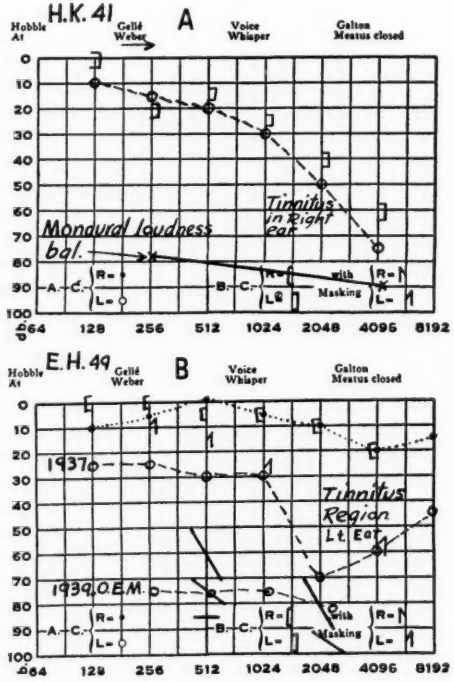

Chart IV. Case A, total deafness in the right ear; possibly a remnant of hearing. Tinnitus in totally deaf ear appears 20 decibels loud. Faint high frequency tinnitus in left ear (5 decibels). Both middle ears appear normal. The AC and BC in the left ear practically coincide. Thermal noise masked the left ear tinnitus at 80 decibels over normal thermal noise threshold. This is 5 to 40 decibels above the threshold level of the frequencies in the tinnitus region. The monaural loudness balance is shown by the straight line near the lower part of the chart. The progressive deafness with frequency and the normal hearing for low tones lead me to believe that the deafness and the tinnitus in the left ear are of peripheral neural origin.

Case B, the hearing in the right ear has always been near normal. In 1937 there were only 30 decibels of deafness in the left ear up to 1024, above which a marked trough occurred in the threshold level, and both the binaural balance test and the bone conduction losses indicated a severe nerve deafness. Two years later (1939) this ear was totally deaf; no sounds of any kind were heard when the opposite ear was completely masked. The plotting on the chart is now only a shadow plotting, owing to insufficient masking. The tinnitus has remained at about the same frequency band and is now heard only in the totally deaf left ear. Neither discrete frequencies nor thermal noise at loud intensities mask the tinnitus. The tinnitus appears to be of neural origin. The deafness and the tinnitus are due to massive lesions. Tinnitus referred to a totally deaf ear in itself suggests a neural origin (stump or central irritation). It cannot be from peripheral stimulation. The diagnonal straight lines at 512 and at 2048 indicate the loudness balance in 1937. They are proof positive that at that time there was hearing in the left ear. Today only a shadow balance can be obtained. It now shows no "recruitment" as in 1937. Both the auditory and the static labyrinths in the right and the left ear in these two patients are now nonfunctioning.

to obtain beats. However, it is difficult to obtain audible beats with very high frequencies. Moreover, very high frequencies (except as a part of noise bands) do not occur in any part of the body with sufficient intensity to be heard. They are damped out. Even with tinnitus in the middle or moderately high frequencies (500 to 4,000) I have been unable to detect true beats although both R. L. Wegel and I have reported two cases of tinnitus with phenomena suggestive of something akin to beats.[3] No certain monaural or binaural beats have been demonstrable to any of my patients without bringing to the ears two frequencies other than the tinnitus, simultaneously applied.

13. This tends to confirm my belief that the biochemical irritations producing tinnitus set up patterns quite different from the normal neural responses to sound vibrations. The fundamental and overtones have no exact representation in non-vibratory tinnitus. The relative activation of the contiguous and separated frequency areas is quite different than with actual pure tones. There is no vibratory interaction to produce summation, difference, interference or combination tones. There are no standing waves. There are no waves of sound whatsoever, only nerve actions and reactions. The biochemical irritation hits all or parts of the acoustic apparatus without acoustic pattern. This is one reason why biochemical tinnitus seldom, if ever, has exactly the timbre of a pure tone. Non-vibratory tinnitus is in fact not a real sound. It is only an illusion of a tone or an illusion of a combination of tones. Nevertheless its timbre and loudness sometimes may be affected by extraneous environmental noise and therefore by changes in external meatal and middle ear resonance, by plus and negative air pressures in the external auditory canal and middle ear, and by closing the external meatus and by changing the size and vibratory characteristics of the ear cavities by vascular congestions or depletion. Environmental noise does not always mask tinnitus. It seems sometimes to accentuate it if it is loud enough to be heard and happens to coincide in frequency with the tinnitus.

14. Experimental neuro-physiology demonstrates that when a nerve is cut the cut end is the source of a continuous stream of impulses, similar to those normally produced physiologically. Enduring sensations like itching, tingling or pain following such lesions can thus be explained. There is a certain synchronization in the automatic discharges from a cut, pressed, or chemically disturbed part of a nerve. Hallowell Davis believes that "mere synchronization of impulses in the auditory nerve would not give a tonal quality to the

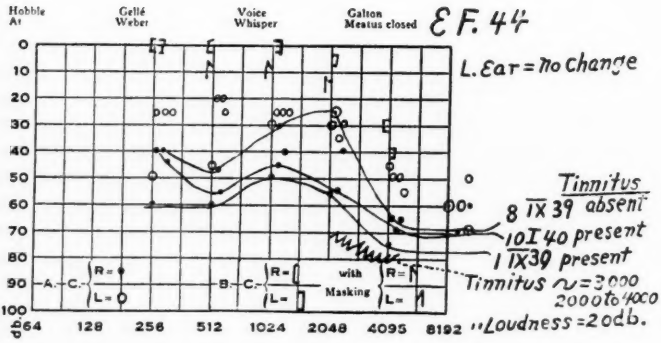

Chart V. Note the great variation in hearing at midfrequencies coincidental with changes in the loudness of the tinnitus, and the small change at low and high frequencies. When the patient is overtired or tense and especially during the menses the tinnitus increases and the hearing diminishes in the right ear, but the hearing in the better left ear changes but little. Audiometric tests show no coincidental change in the left ear. Only the extreme swings in plotting for the right ear are shown by the continuous lines. The bone conduction at frequencies 1024 and 2048 varied 10 to 15 decibels, with twice these changes in air conduction. Thermal noise masked the tinnitus at 15 decibels above the thermal noise threshold. Although the tinnitus is in the region of 3000 (along the slope of the dip), the hearing change extends over several octaves below this frequency region. This is a frequent observation in several types of audiogram. It would appear that all parts of the lesion causing the deafness are not necessarily accountable for the tinnitus and that tinnitus and deafness do not necessarily correspond in frequency band or in degree. This tinnitus would appear to be neuroperipheral in origin.

resulting sensation"; that "electrical stimulation of the auditory nerve in man gives rise only to a sensation of noise unless the cochlea is intact and presumably set in vibration"; and that "many other lines of evidence also show that the frequency theory of hearing is untenable."

If Davis is right, tonal quality depends only upon the mechanical activation of a group of neural elements enervating a particular section of the basilar membrane and not to a tendency of the neural elements here or elsewhere to synchronize their discharges. But the fibers from each area of the basilar membrane are spatially implicated as are their central distributions. Whether or not Davis is right, spatial representation in end organ or nerve does not mitigate against the hypothesis that there can be central as well as peripheral origins for tinnitus.

It must always be kept in mind that most electrical stimuli affect masses of neural elements and consequently produce a mass effect, a noise, not a discrete tone, whereas biochemical lesions may be more localized and may therefore activate areas associated with discrete frequency perception.

It is also conceivable (but difficult to prove) that tonal tinnitus may be the result of closely synchronized discharges from contiguous areas in the organ of hearing, and that the resulting tinnitus may be due to the beats between their two differing rates of synchronized discharges. The set-up might be something like that from two sets of closely synchronized audio frequency discharges. Any frequency can be produced by such arrangements as is done in the audiometer.

15. I have shown that tinnitus may be very difficult to mask even when it is faint (even when only 5 to 10 decibels over threshold loudness as compared to a normal ear) so that the loudness of a tinnitus and the difficulty in masking it do not always run parallel. What plausible reason can be given for such a departure from the expected phenomena? Is it that in such instances the difficulty in masking is caused only by a lack of neural elements (hair cells) in the particular section of the organ of Corti which would normally be activated by the applied masking sound. In other words, is it because there are insufficient or no sensory elements available to respond to the masking noise or tone in or near the frequency band affected by the lesions causing the tinnitus? If this is so the true explanation would appear to be purely the difficulty or impossibility of submerging or masking the tinnitus in even a widely spread or high level of vibratory stimulation. But this would appear to ignore

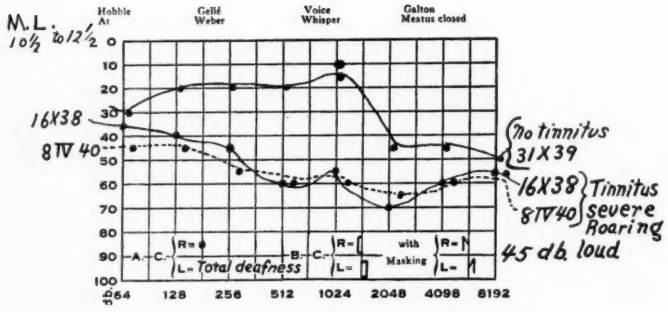

Chart VI. At my first examination there was a loud tinnitus (45 to 50 decibels) in the right ear, like a loud thermal noise. There was no tinnitus in the totally deaf left ear. Following bilateral parotitis and meningitis four years ago there was transitory loud tinnitus in both ears, followed by deafness, vertigo and nausea. The vertigo and nausea persisted two months with marked loss in hearing in the right ear and total deafness in the left ear. When speech hearing in the right ear improves 40 decibels by air conduction and bone conduction at midfrequencies, the tinnitus in the right ear ceases. When the hearing goes down, the tinnitus returns. This I have observed many times. It is equivalent to a change in hearing from 1 foot (0.3 meter) to 20 feet (6 meters). Although one and a half years elapsed between the extreme low periods, there is little difference between these lows at the different frequencies. The bone conduction varied with the air conduction at all frequencies, but when hearing was up, bone conduction at 128 was normal and at 250 was —10 decibels. No other changes were noted in the otologic picture. Thermal noise and air noise masked the tinnitus at 10 decibels over their thresholds. On account of the nerve deafness this is a very loud sound. By actual measurment it is equivalent to about 40 decibels in a normal ear. The patient thinks she "feels" the tinnitus in spite of the masking noise. Note that whether or not the tinnitus was present the hearing above 4000 varied little. These high frequencies are above those most important for speech, and therefore this lack of change did not prevent great improvement in "speech hearing." All this suggests that the tinnitus is nonvibratory and that both the tinnitus and the variations in deafness are vasomotor in origin, both the peripheral and the central auditory neural apparatus being involved.

the recruitment phenomenon. In other words, it would suggest that no recruitment is present in the presence of tinnitus. I have repeatedly shown the reverse to be true, that even in an ear with severe tinnitus a very loud sound in the tinnitus band of frequencies will appear as loud, or nearly as loud, as in an opposite normal ear (Chart IV). Therefore a mere unavailability of neural elements because of tinnitus does not prevent the ear from being masked by loud sounds. In fact such an ear is submergible to about the same extent as a normal ear.

If the neural elements are entirely absent they cannot be activated by any irritation, in which case there will be no evidence of life in them; no tinnitus can emanate from them. Tinnitus is an evidence of life. The inevitable presence of the recruitment phenomenon in the presence of tinnitus would seem to preclude a failure in the cochlear neural elements to pick up and recruit and transmit sufficient impulses to block out a sensation of tinnitus arising in the cochlea, but may be not from a tinnitus arising centrally. I therefore suggested that the difficulty of masking may be due largely to a lesion, medial or central to the end organ, and that the origin of the tinnitus in such cases was medial or central to the end organ, at least in large part. If this is the true explanation there is at hand a ready test for differentiating these two types of non-vibratory tinnitus: peripheral (cochlea), medial (cochlear nerve), and central (nuclei and central pathways).*

16. When tinnitus is bilaterally equal in pitch and loudness, is it of central or peripheral origin? In other words, can two ears be pathologically, coincidentally, bilaterally equal in the areas producing the tinnitus, as they can be closely bilaterally equal in audiograms? Hallowell Davis is of the opinion that "There is no exact analogy to my theory of central tinnitus in other sensory modalities", and he is therefore "hesitant to accept the possibility of a central unilateral or bilateral tinnitus as a common phenomenon." Moreover, to do so would seem to him to imply some rather definite focal lesions which he would not expect the auditory mechanism to be uniquely subjected to.

In opposition to this opinion I call attention to the fact that although both of our ears have the same general hereditary background they also have hereditary differences, and that their internal

*In peripheral tinnitus the afferent neural pathways from the cochlea are available for masking impulses. In medial and central tinnitus part of the afferent neural pathways from the cochlea to the cortex are not available for masking impulses. They are blocked off en masse.

and external environments usually differ greatly at times. Although their hearing threshold curves may be similar in general design some differences always occur. I also call attention to the occurrence of tumors and other pressure lesions to which the central pathways of the cochlear nerves are unilaterally or bilaterally subjected, i.e., multiple sclerosis, arteriosclerotic plaques, local vascular congestions, hydrostatic imbalance, brain stem tumors, etc., and also to the frequent cessation of tinnitus even when only the vestibular nerve is sectioned and the pressure on the cochlear nerve relieved. Atrophy and lessened local vascular tissue make room for (decompress) the cochlear nerve. Dr. R. Lorente de Nó agrees with the hypothesis that central lesions can and do cause tinnitus.

It should be kept in mind that tinnitus similar in tone if bilaterally unequal in intensity will tend to be heard solely in one ear, on the louder side. Its bilateralness can then be detected only by masking out the louder side. Environmental noise as well as the contra-lateral tinnitus may easily mask out the weaker side. I believe the lesion is probably in the cochlea if environmental noise masks tinnitus with ease. If loud noise does not mask faint tinnitus I suspect that the lesion is in large part central to the end organ.

17. Some fifteen years ago I first called attention to the association of tinnitus with dips and troughs in the audiogram threshold curve of hearing.[5] The tinnitus was interpreted as representing an irritation of the hearing end-organ elements, which is preliminary to subsequent degeneration. It is then, if sustained, a threat or danger signal of increasing deafness ahead. It is a definite sign of potential or impending deafness. These observations are in accord with the discovery that the pitch of tinnitus often does not correspond to the bottom of the trough, at the depression in the audiogram, at the point representing the greatest neural degeneration. It usually occurs in the frequency areas near the lower or the upper brows of the slopes (near the brink of the hill) where its more normal and subnormal areas blend. Also it has been observed the deeper the trough the less often does the tinnitus frequency correspond with its deepest part. These observations correlate with those in other fields of sensory perception. It is a well established law of physiology that sensation is limited to the area of greatest stimulation. It is at this point where there occurs the greatest difference in environment.

As a biochemical analogy between one tactile sensory modality and another, note the following: One usually refers cutaneous itching, burning, or pressure sensations to the periphery of a stimulated or injured area of integument. For touch and pressure this is due

to the nature of the stimulus, i.e., a deformation and mechanical stretching of the tissue and its contained end organs, in the case of itching, from a persistent neural discharge from zones of slight or moderate damage and not from the more severely damaged or degenerated tissues.

Peripheral non-vibratory tinnitus may therefore be likened to an irritation (tingling, tickling, or itching) of the basilar membrane and its tonal quality due to irritation on particular neural areas of the membrane. If the irritation is sufficiently extensive, the tickling will appear to the patient more like a jumble of sounds the result of a wider band of frequencies: a roaring or hissing (Chart VI).

Medial or central tinnitus on the other hand may be said to represent a less differentiated, mass irritation of neural elements. The tinnitus in such mass irritations shows little if any resemblance to patterns obtained from the reception of actual sound, except thermal noise which it closely resembles. Like thermal noise it is made up of all frequencies jumbled together. It can result only from a mass or block stimulation. The tinnitus experienced as a part of Ménière's symptom complex is usually of this character. The sensations are due to the rhythmic or more or less synchronized firings of a very broad band of neural elements, irritated by pressure, degeneration, or other biochemical irritations, in the same way that other nerves of sensation may be activated under comparable circumstances.

I have been unable to learn of anyone who has demonstrated non-vibratory tinnitus in aural microphonics. Experimental neurophysiologists tell me they would not expect to find tinnitus in the electrical outfit of the cochlea, except in the presence of actual mechanical vibrations, within or brought to the inner ear. One reason is the ever-present background of physiological activity, muscle sounds, and unavoidable amplifier noise. Aural microphonics appear to be the result of a piezo-electric effect in the hair cells in the same way as I suggested that the nerve impulses from the maculae of the utricle and the sacule also were probably piezo-electric in origin from torsion of the otolith organs,[5] a transformer effect of changing mechanical torsion and pressure into electrical energy (nerve impulse).

SUMMARY AND CONCLUSIONS

1. I have divided tinnitus into two fundamental types, vibratory and non-vibratory. The first is mechanical in origin due to actual vibrations. The second is biochemical in origin and not due to any actual vibration.

2. It is shown how the loudness of tinnitus may be objectively estimated by comparing it to an applied tone or noise of known loudness. The loudness balance technique used for this purpose is described and discussed.

3. Attention is called to the "illusion of great intensity" of tinnitus. In many instances although the patient says his head noises are so loud as to be unbearable, they are actually very faint, as determined by the loudness balance technique.

4. Methods for estimating the tone or frequency band of tinnitus are discussed.

5. The maskability of tinnitus by discrete and by band frequencies (thermal noise) is shown graphically with audiograms of different types of hearing loss. It is shown conclusively that in some instances the tinnitus is very easily masked at all frequencies. In others the masking is greatest as the masking tone approaches near to the frequency band of the tinnitus. In others the masking is difficult, and in some it cannot be accomplished even by using the loudest masking sounds. It is pointed out that non-vibratory tinnitus often sounds like escaping steam or air, or the sibilant "sh". Since this is exactly like the sound of a thermal noise covering all frequencies up to 10,000 in about equal intensity, it is suggested that hissing tinnitus is carried by the simultaneous irritation of a very large number of neural elements.

6. It is demonstrated conclusively that neither the loudness of the tinnitus nor its frequency alone determine the ease of masking it. The implications of these findings are discussed at some length.

7. It has been found that the presence of tinnitus is always associated with more or less deafness. The pathophysiological implications of this finding are discussed.

8. It is pointed out that the frequency and loudness of tinnitus determine the deafness caused by the tinnitus lesions. This does not necessarily coincide with the deafness in general or the fluctuations of "speech hearing".

9. The factor of fatigue from tinnitus or irritations causing tinnitus is discussed.

10. The mechanism of the production of tinnitus is discussed in the light of neurophysiological research, and the absence of non-vibratory tinnitus in cochlear microphonics is pointed out.

11. Attention is called to the fact that increases in the loudness of tinnitus are often attended by an elevation in tone and vice versa. A probable explanation of this phenomenon is discussed.

12. Attention is called to the fact that it has been impossible to demonstrate beats between non-vibratory tinnitus and applied pure tone, and that only actual vibrations (not biochemical) can be heard by someone other than the subject.

13. Biochemical irritations do not follow acoustic patterns, and this is one reason why non-vibratory tinnitus seldom if ever has the timbre of a pure tone. It is in fact only an "illusion of a tone" or an illusion of a combination of tones.

14. That neurophysiology and pathology have a bearing on tinnitus is discussed.

15. It would appear that the difficulty in some instances in submerging or masking tinnitus cannot be explained wholly on the theory that there are insufficient or no sensory elements available in the peripheral apparatus to respond to the masking sound because the "recruitment phenomenon" would function as long as there were any available neural elements left, and this would make it possible to mask out the tinnitus, particularly if it were peripheral in origin.

It is pointed out that tinnitus is an evidence of life and that where there is tinnitus there is also present the "recruitment phenomenon". It is suggested that the difficulty in masking may be due largely to a medial lesion or one central to the end organ, and the origin of the tinnitus in such cases is medial or central to the end organ. If this is the true explanation, we have at hand a test for differentiating the types of non-vibratory tinnitus, peripheral, medial, and central.

16. The mechanism of bilateral tinnitus, of peripheral and central origin, is discussed.

17. Tinnitus, if sustained, is a definite sign of increasing or potential deafness.

The correlation of all the observations with those in other fields of sensory perception is discussed, particularly the association of tinnitus with dips and troughs in the audiogram curve. The tinnitus is interpreted as representing an early irritation preliminary to subsequent degeneration. Biochemical analogies between different tactile sensory modalities are pointed out, and peripheral non-vibratory tinnitus is likened to a tickling, tingling, or itching on particular

areas of the basilar membrane. Medial or central tinnitus represents a mass irritation of neural elements with little or no resemblance to the patterns obtained from an actual sound except a thermal noise.

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XIII

CHEMOTHERAPY IN THE TREATMENT OF COMPLICATIONS OF ACUTE MIDDLE EAR SUPPURATION*

(Petrositis and Meningitis)

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The treatment of otitic complications has undergone two radical and distinct improvements in the past few years.

The first of these came with the development of adequate methods for diagnosis and surgical treatment of suppuration in the deeper parts of the temporal bone. On the basis of accurate pathological, anatomical and X-ray studies surgical methods were developed to reach foci of suppuration in all areas of the pyramid as well as in the mastoid. It became possible to prevent mortality from complications of acute suppuration in nearly all cases where the infecting organism still remained outside the barrier of the dura at the time proper surgical intervention was carried out.

At about the same time that these developments in diagnosis and surgical care were becoming more widely understood, this advance in our knowledge was overshadowed by the second great improvement in therapeutic methods, which came with the advent of sulfanilamide.

Four years of therapy with sulfanilamide and two years with sulfapyridine have resulted in numerous successes in the treatment of the one complication which had previously been almost one hundred per cent fatal, diffuse suppurative leptomeningitis. There is also evidence that the widespread use of these drugs has reduced the incidence of surgical mastoiditis.

The experience at the University of Chicago Clinics during the past three years has been that the number of operations necessary for acute mastoid suppuration has dropped materially, which apparently corresponds with general experience throughout the country.

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*From the Division of Otolaryngology of the University of Chicago.

Presented before the Chicago Laryngological and Otological Society, Monday, November 4, 1940.

The incidence of complications of acute suppuration of the middle ear and mastoid does not appear to have fallen in the same ratio. The total number of complications treated in the season of 1939-40 in this clinic was greater than in any preceding year. This particular experience probably does not reflect the general situation as to the incidence of complications, but it does definitely indicate that the problem of otitic complications has not yet been completely solved.

Reports of a statistical nature afford some general indication as to the effect of the therapy on the incidence of disease, but further improvement in therapy both chemical and surgical requires accurate analysis of individual clinical cases.

With the object of determining more closely the causes for success and failure and thereby contributing to the establishment of criteria for successful therapy, an analysis of a group of clinical cases has been made.

This group consists of two types of complication of acute middle ear suppuration, namely, petrositis, and meningitis. All cases were treated during 1939-40 with the exception of one treated in 1938 and previously unreported, which contributes to the subject under investigation.

SUPPURATION IN THE PETROUS PYRAMID

Five cases of acute suppuration in the petrous pyramid were treated during the past fall and winter season. Three of these had meningitis and are discussed under that heading. One additional case of petrositis and meningitis treated in the early part of 1939 is included under meningitis since it contributes pertinent evidence on the question of therapy.

Two cases of petrositis without meningitis are described here.

REPORT OF CASES

CASE 1.—T. W., a steel worker, 38 years old, gave a history that one year previously he developed pain and deafness in the right ear, followed by "Bezold's abscess", and operation. His hearing was reduced to 10 per cent before operation and was totally absent after. One year later, August 19, 1939, there was an onset of left-sided earache after coughing, followed by deafness and pains in the left side of the head. He was seen in consultation with Dr. H. Kobrak September 26, 1939. Serous fluid was liberated from the middle ear on myringotomy. Pneumococcus Type III was cultured. The ear was dry in three days. Myringotomy was repeated by Dr. Kobrak. The patient complained daily of pains in the left occipi-

tal and parietal regions and in both eyes. There was a scant serous discharge. On October 2, 1939, he began to have vertigo. Dizziness increased daily, and hearing steadily diminished. He was referred for admission on October 7, 1939, with hearing so reduced that only a loud shout could be interpreted at the ear. Bone conduction was markedly reduced. There was spontaneous nystagmus third degree to the left. No hearing was demonstrable in the right ear.

X-ray of the pyramids was complicated because of previous disease in the right pyramid. The presence of pneumatization in the superior perilabyrinthine region was indicated on Stenver's view with the possibility of a break in the cortex visible on the occipital view.

The diagnosis was of an acute mastoiditis, left, with serous labyrinthitis and posterior petrositis (perilabyrinthine suppuration). A complete mastoidectomy was done without finding sufficient cause for the labyrinthitis and deep seated headache. The retrofacial cells were extensive but stopped at the jugular bulb. After skeletonizing the labyrinth a channel was made through the superior angle, where the bone was softer than the labyrinth capsule, but without a visible tract. When the channel in the angle was excavated beyond the level of the common crus of the superior and posterior canals, the bone became softer and a large abscess was opened. A soft rubber drain was left leading into the abscess. The nystagmus and vertigo promptly decreased and were absent in four days. Hearing returned more slowly. In one month he had good hearing for conversation, and the hearing threshold at 1000 cycles was 25 db. compared with 80 db. at time of operation. He suffered mild headaches at times for several months. Healing was rapid.

Comment: Surgical drainage of the focus of suppuration in the pyramid gave a successful result. In retrospect however it must be realized that this patient was in imminent danger of losing the function of the ear and of developing a meningitis. The complication in this type of case sometimes develops swiftly as in Cases 8 and 9 of this report. Surgical treatment is adequate in such cases provided it is carried out before the labyrinth is destroyed or the meninges invaded.

Chemotherapy could not, in the light of present knowledge, have been expected to result in a cure after the suppurative focus had developed, without surgery. Two questions arise however: First, would heavy dosage of the drug in early stages of the otitis have prevented the necessity of surgery? Second, when confronted with urgent surgical indications, should not chemotherapy be administered as the initial step in treatment?

CASE 2 (Chart I).—H. M., male, aged 26, was admitted with scarlet fever February 2, 1940. There was an onset of right otitis media the same night. Chart I shows the dosage of sulfanilamide throughout his illness. He was referred to the otolaryngological service February 21, 1940. There was a profuse purulent discharge from the right ear. X-ray showed extensively pneumatized petrous pyramids with diffuse clouding of the right mastoid, perilabyrinthine and apex cells. An attempt was made to find out what effect heavy dosage of sulfanilamide might have on the otitic suppuration. After one week of heavy dosage there was no appreciable decrease in discharge. The temperature rose moderately, and the patient had a chill. A complete mastoidectomy was done. Sulfanilamide was again used in heavy concentration. The discharge continued profusely. The patient had intermittent headache on the right side, mostly parietal and frontal, which was relieved by aspirin. Right sixth nerve palsy developed and became complete in a

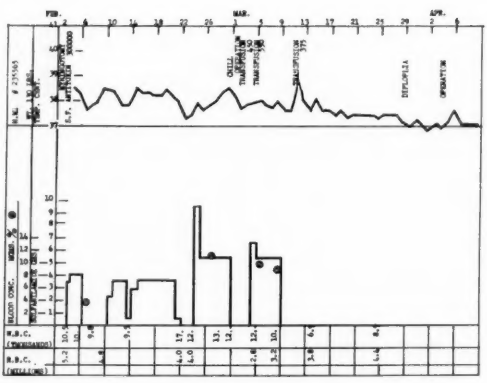

Chart I—Case 2. Scarlet fever otitic suppuration. Extensive development of middle ear air spaces. Sulfanilamide ineffective. Large abscess in petrous apex drained by the posterior route. Recovered.

few days. C.S.F. was clear. There was no sign of meningitis. The mastoid was reopened and a tract made by means of curettes through the angle, above the internal meatus, into the apex, where a large abscess was opened (Fig. 1). The distance from the common crus of the vertical canals down into the bottom of the cavity in the apex measured 5 cm. Diplopia disappeared in four days. Recovery was uneventful and the ear dry in ten days.

Comment: High concentration of sulfanilamide was ineffective, after suppuration had become established. Unusually extensive pneumatization of the temporal bone undoubtedly was a factor in the failure of chemotherapy. The possibility must be admitted that higher concentration of the drug within the first few days before there was breakdown of bone might have been more effective, but this is doubtful. The moderate dosage of the drug in the early period of disease produced no obvious change in suppuration.

The case may be classed as posterior petrositis, although the chief collection of pus was in the apex. The indications for surgery were clear and the result successful, with preservation of the labyrinth and middle ear apparatus.

DIFFUSE SUPPURATIVE LEPTOMENINGITIS

Seven cases of diffuse suppurative meningitis with positive cultures and one of basilar meningitis, secondary to acute middle ear suppuration, are reported. There were two fatalities.

Case 3.—C. P., aged six, was admitted March 8, 1938, with diffuse hemolytic Streptococcus meningitis, following scarlet fever, lobar pneumonia, and right acute otitis media of over six weeks' duration.

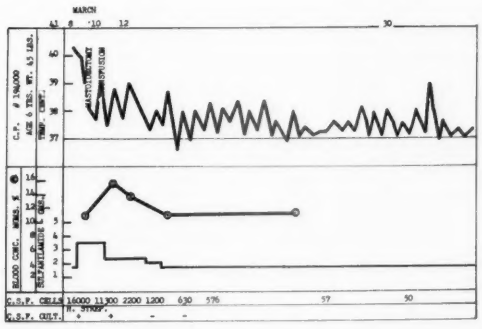

Chart II—Case 3. Menginitis following post-scarlet fever otitis media and mastoiditis. Recovery.

Chart II shows the course after admission. At the mastoid operation, the cells were filled with gray swollen mucosa but little purulent secretion. There was no X-ray evidence of petrositis. There was complete right sixth nerve palsy.

Blood concentration of sulfanilamide reached 16.2 on the third day and was maintained above 10 mg. per 100 cc. until the spinal fluid cell count was nearly normal. The child made a complete recovery.

Comment: Since no definite collection of pus was found at operation it seems that surgery may have had little influence on the recovery. However the clinical course indicated mastoid suppuration as the source of meningeal invasion. A focus of necrosis or abscess tends to be resistant to chemotherapy, and the only means of promptly excluding such a probability is by mastoid surgery.

Case 4. (Chart III). H.G. male, eight years old, was admitted February 26, 1940 with diffuse meningitis, following right middle ear suppuration of ten days' duration. The right mastoid had been operated on February 23.

Intravenous sodium sulfapyridine gm. 2.0 was given on admission since the organism was unknown and child was vomiting. Hemolytic streptococci were cultured from the C.S.F. On February 29, 1940 sulfanilamide by mouth was started and tolerated well. Chart III shows the course of the illness. Points of significance were that a lowering of the blood concentration to 6.8 mg. per cent at the end of one week was followed by an exacerbation of the meningitis. Mastoid revision on March 5, 1940 revealed no significant focus of suppuration. At the end of two weeks, with C.S.F. concentration of 7.0 mg. per cent, the C.S.F. culture was again positive and cell count 252.

Blood concentration was not determined after March 9, 1940 because of the difficulty in entering the veins. The C.S.F. concentration was maintained until March 21, 1940 because of persisting fever and spasticity. A rash appeared March 22, 1940. The temperature elevation after March 23, 1940 was explained by an upper respiratory infection.

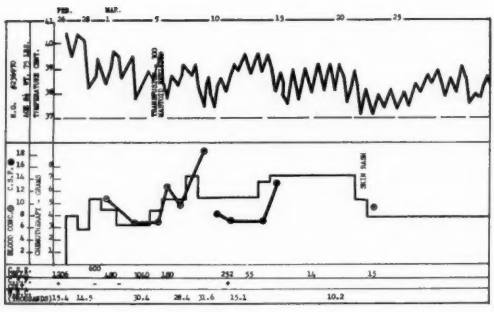

Chart III—Case 4. Hem. Strep. meningitis following otitis media and mastoiditis. Exacerbation at end of one week when blood concentration fell below 7 mgm. per cent. Recovery.

Comment: The concentration of the drug during the first week fell below what was necessary to control meningitis. Both here and in Case 7, failure to control the disease at the onset appeared to result in a prolonged illness, during which very high dosage had to be maintained over a long period to effect a cure.

Heavy dosage at the onset, to produce and maintain the blood level above 14 for five to seven days or longer if necessary has given better results (Cases 3, 8, 5).

Failure to control the meningitis completely in early stages probably allows accumulation of purulent exudate in the cisternae at the base of the brain which tend to resist chemotherapy. Concentration (over 18 in the blood and 13 in spinal fluid) was maintained without injurious effects.

It is probable that recovery was not affected by the mastoid revision and exenteration and possibly not by the original antrotomy since no definite focus of necrosis was found. However the history indicated strongly a mastoid origin for the meningitis. The unfavorable condition of the patient and the possibility of a persisting focus of necrosis appeared to indicate surgery as the safest course to follow.

Case 5 (Chart IV).—T. E. male, aged 11 years, was admitted February 10, 1940 with an acute mastoid suppuration, left, with severe intermittent left frontal headache and high temperature. Hemolytic streptococcus was cultured. Following an onset of middle ear suppuration two weeks previously he had been hospitalized for ten days and discharged with the otitis apparently subsided. The child had had a bilateral mastoid operation at the age of six years.

X-rays on February 10, 1940 showed pneumatization in the superior perilabyrinthine region and also in the apex bilateral (see Fig. 2) with left side clouded. Complete mastoidectomy and elevation of the dura beyond the arcuate eminence were negative and followed by no improvement in symptoms. Sulfanilamide was started by mouth but was vomited each time. Only 1.8 gm. was re-

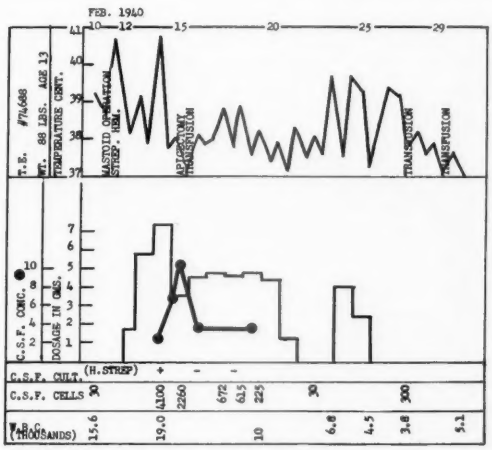

Chart IV—Case 5. Extension to the meninges from the petrous apex. Hem. Strep. In this case as in others many organisms were found on smears of centrifuged spinal fluid, but cultures were negative after saturation with the drug. Sodium sulfapyridine intravenously was used after Feb. 14.

tained the day after operation. Intramuscular injection of prontosil was added the following day with a total dose of 5.8 gm. Sodium sulfapyridine was begun intravenously February 14. Meningitis developed after the operation, and on February 14 the C.S.F. cell count was 4100. Gram positive diplococci and chains were found on culture. Many organisms were seen on the smear.

On February 15, 1940 the petrous apex was opened up via the middle ear, eustachian tube and carotid canal and explored thoroughly. No pus could be recognized. The course of the illness is shown in Chart IV.

Comment: The persistent unilateral frontal headache and development of meningitis after thorough mastoid exenteration including elevation of the dura above and behind the labyrinth indicated the clouded petrous apex as the probable site of extension. Admittedly there might have been recovery without the petrous apex operation, but with such definite indications the safest course was to provide surgical drainage.

The absence of a gross effusion of pus, sufficient to well up into the wound, is not uncommon. A focus of necrosis which has partial drainage through a fistula to the exterior, or into the meninges, may contain much infected granulation tissue with small scattered necrotic areas and abcesses, but surgical drainage may reveal no macroscopic collection of pus. This has been demonstrated by the author both clinically and on histopathological examination. (1, 2.)

Recurrence of fever and increased C.S.F. cell count after withdrawal of sulfapyridine occurred in this and several other cases. Hypochromic anemia and

depression of granulocytes at the same time presented a problem in therapy since these blood changes may have been due to the drug rather than infection.

CASE 6.—J. D., male, age about six years old, was seen in consultation with Dr. George Livingstone about ten days after a right mastoidectomy had been done. There was persistence of irregular spikes of fever, irritability, extremely profuse discharge of pus from the ear, increasing W.B.C., and intermittent frontal headache. Hemolytic streptococcus was found on mastoid culture.

X-rays showed well developed cells in the superior perilabyrinthine region on the normal side, with evidence of a break in the cortex medial to the arcuate eminence on the diseased side (Fig. 3). On the axial view the apexes appeared to be nonpneumatic.

The mastoid operation was revised. After removal of cells as far as the capsule of the superior canal, the possibility of a cell connection from the mastoid to the cells beyond the arcuate eminence was ruled out. The dura was therefore elevated over the arcuate eminence, and just beyond an extradural collection of pus was drained. The break in the cortex could be felt with a blunt hook. Several cubic centimeters of pus were liberated and a drain left in position. A spinal puncture was not done previous to operation. The child continued to vomit after operation and signs of diffuse meningitis were present. He was treated by sulfanilamide under Dr. Livingston's direction and made a complete recovery. The pathology corresponded to that previously described by the author.[1]

Comment: Sulfanilamide must be credited with the cure of the diffuse suppurative meningitis.

Drainage of the extradural abscess resulted in prompt disappearance of the extremely profuse aural discharge and undoubtedly removed the source of the meningitis, but unfortunately not until the barrier of the dura had been broken through.

Inasmuch as we have definite knowledge that the drug may be ineffective in the presence of a collection of pus such as was present in this case it is imperative that the focus be drained to prevent reinfection of the meningeal spaces.

CASE 7.—A boy, aged 7, was treated by Dr. Meyer Fox, Milwaukee, Wisconsin, for hemolytic streptococcus meningitis. This patient was ill for seven days, with discharging ears for three days before admission. (See Chart V.) Nine days after admission diffuse meningitis had developed although he had been treated by moderate dosage of neoprontosil. A mastoid operation followed by higher dosage of sulfanilamide and neoprontosil brought the meningitis under control, but ten days later there was another exacerbation. At this time the patient was seen in consultation regarding the possibility of a persisting focus in the petrous pyramid. X-rays of the pyramid were negative, and the possibility of a persisting focus in the bone or extradurally was ruled out by a revision of the mastoid. The exacerbation of the meningitis was interpreted as the result of too early withdrawal of the drug. High dosage was then started and maintained for 11 days, then gradually reduced.

Comment: Several points are illustrated by this case.

1. Surgery was of doubtful significance in the recovery of this patient. The small-celled type of mastoid, of limited extent, responds better to chemotherapy

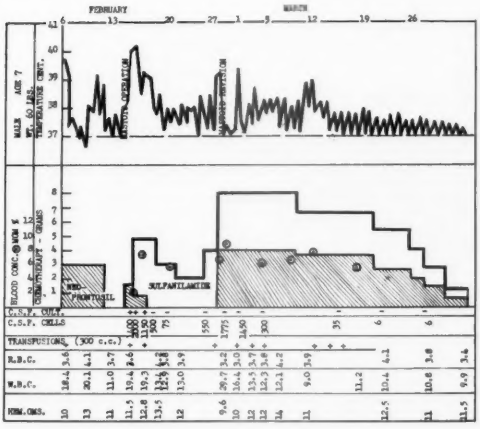

Chart V—Case 7. Blood concentrations are uniformly low in relation to the dosage. Premature lowering of the dosage during week of Feb. 20th was followed by exacerbation of the meningitis. Hem. Strep. meningitis with recovery.

than the large, extensively pneumatized type. But such possibilities as extradural or subdural abscess cannot be excluded without surgery.

2. As was shown by Case 4 chemotherapy seemed to be less effective as the disease became more prolonged. Possible explanations for this would be a state of symbiosis developing between the organism and the therapeutic agent or the tendency for pocketing or development of localized foci in certain areas, for instance around the base of the brain. High concentrations (over 14 mgm. per cent) maintained for the first four to seven days of therapy probably obviate these possibilities.

3. Blood concentrations were uniformly much lower than are usually obtained by similar dosage, which probably explains the persistence of the disease.

The intravenous injection of sodium sulfapyridine may be substituted with success under such circumstances, as was done in Case 5.

Case 8.—N. W., an adult female, was admitted May 2, 1940, with bilateral acute middle ear suppuration following a cold of two weeks' duration. She was hospitalized for six days. Pneumococcus Type V was found on culture.

The day after admission sulfanilamide was started, and the following day after learning the culture report, sulfapyridine was substituted. Sulfanilamide gm. 7.2 and sulfapyridine gm. 11.0 were given in three and a half days.

Two days later patient left the hospital, having had normal temperature for four days, with both ears dry.

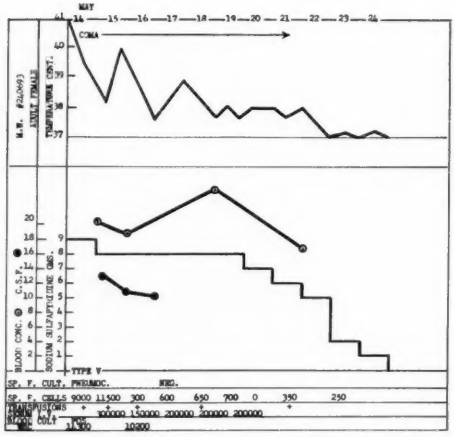

Chart VI—Case 8. Pneumococcus Type V meningitis, secondary to petrositis. Recovery.

Six days later, May 14, 1940, she was admitted with diffuse suppurative meningitis and the right ear discharging profusely again. There had been an onset of severe frontal headache the night before and vomiting the morning of admission. Pneumococcus Type V was found on spinal fluid and blood cultures.

The patient received 5 gm. of sodium sulfapyridine intravenously on admission, 9 gm. altogether on the first day and 8 gm. daily for five succeeding days. Chart VI shows the record of temperature and therapy. Patient was given antipneumococcus serum but, due to a misinterpreted report by telephone, did not receive the correct type of serum until the second day. Fluids and medication were all given intravenously. Fluid intake was maintained between 3000 and 4000 cc. daily and calcium nembutal given for sedation. The patient was comatose for six days. Intrathecal injection of 13 cc. of the patient's serum filtrate were given on the third day and from 10 to 15 cc. on the following three days.

On the day of admission X-ray showed clouded mastoid cells on both sides and a large cell in the angle of the pyramid adjacent to the internal meatus bilateral, which was clouded on both sides but more dense on the left.

In view of the active suppuration on the right side, the right mastoid was cleaned out and a tract made through the angle of the pyramid by Dr. H. B. Perlman until the curette dropped into the large cell. A gush of bloody exudate welled up in the tract. A probe was placed in the cell and a picture made with the portable X-ray machine (Fig. 4).

Comment: The apparent cure of the otitic suppuration in the initial period in hospital illustrates the "masking effect" of the drug which may lead to a false sense of security.

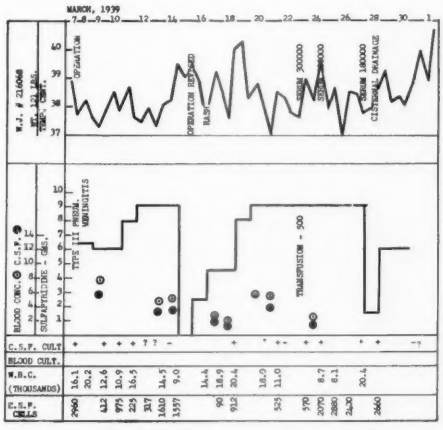

Chart VII—Case 9. Pneumococcus Type III meningitis secondary to petrositis. Died.

In view of the rather mild pathology in the mastoid the meningeal extension undoubtedly occurred from the cells in the pyramid. Knowing the tendency for a focus of suppuration to resist chemotherapy, adequate surgical drainage was indicated.

The relative effect of sulfapyridine and serum therapy in the spinal fluid seems clear in this case. The C.S.F. cell count had already dropped to 300 when the first intrathecal injection was made. The agglutination titre for Type V pneumococcus on the fourth day was zero, and by the fifth day, 1 to 4. In such desperate situations however all therapeutic measures should be utilized, namely, chemotherapy, surgery, type-specific serum and transfusions.

CASE 9 (Chart VII).—W. J., male adult, was admitted March 7, 1939, with Pneumococcus Type III meningitis, following otitis media, right, of five weeks' duration. The ear had been dry for two weeks. There was a persistent right-sided headache. He was seen the day before admission as an ambulatory patient. X-rays were taken but patient decided to return home. There was a sudden onset of vomiting during the night.

X-rays showed clouded mastoid cells and on the occipital view a large tract of cells extending as far as the internal meatus bilaterally, clouded on the right. (These X-rays are illustrated in a former report,[2] Fig. 7C.)

The mastoid was exenterated and the abscess in the pyramid drained by the posterior route. Cerebrospinal fluid came away with the pus.

Comment: The meningitis was a direct extension of petrous pyramid (perilabyrinthine) suppuration. This case illustrates the important fact that headache

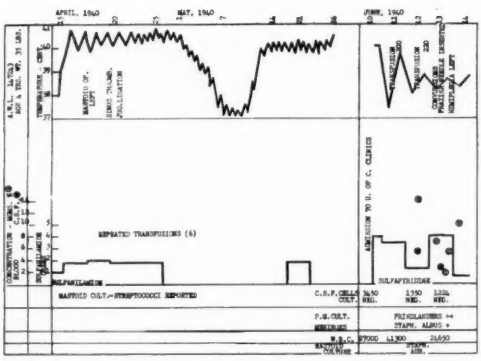

Chart VIII—Case 10. Subdural, subarachnoid and small adjacent cerebellar abscess secondary to acute mastoid suppuration and sinus thrombosis.

in the presence of an otitis media must be interpreted as a danger signal and that the onset of meningitis may be sudden.

Such a case as this may be accurately diagnosed and cured by surgical treatment if the barrier of the dura has not been already crossed by the infection.

After a diffuse meningitis is established, all available therapeutic methods may fail.

High dosage with sulfapyridine failed to produce a high concentration in the blood and spinal fluid. In Case 7 a C.S.F. concentration nearly three times as great was maintained by comparable dosage. The fluid intake was maintained at approximately the same levels in both cases, from 3000 to 4500 cc. daily.

Case 10 (Chart VIII).—A. W. L., male, aged four, was admitted June 10, 1940, semicomatose, with papilledema and definite meningeal signs. There had been an onset of middle ear suppuration two months earlier, with high fever. Mastoidectomy was performed by Dr. M. Hipskind, and several days later ligation of the internal jugular and removal of an infected thrombus from the sigmoid and transverse sinuses were carried out. Streptococcus was reported on the culture from the mastoid. A record of the temperature and sulfanilamide therapy up to May 26, 1940, was supplied by Dr. Hipskind. C.S.F. cultures after admission June 10, 1940, were sterile. X-rays failed to show any pneumatization of the petrous pyramids. The coma increased, and four days later quadraplegic spasm developed. A Frazier needle was inserted in the right parietal region by Dr. E. A. Walker and over 60 cc. of clear fluid removed. Left hemiplegia developed on the following day. The patient expired.

The postmortem examination showed a large collection of pus between the left cerebellar hemisphere and the falx, with an area of thick exudate in the subdural and subarachnoid spaces bordering on the posteromedial surface of the petrous

pyramid. There was a small necrotic abscess 6 mm. in diameter in the adjacent cerebellar hemisphere, which was confined to the folia and did not penetrate the white matter. There was basilar meningitis but none over the convexity of the brain.

Friedlander's bacillus was obtained on postmortem cultures from both the upper and basilar meninges. An organized clot was found in the left lateral sinus, and a recent red thrombus in the right.

Serial sections of the temporal bone showed the collection of pus in the subdural and subarachnoid spaces, with much swelling of the meninges and granulation tissue formation. There was no suppuration in the pyramid (confirming the X-ray impression) and no extradural collection of pus. The abscess in the cerebellopontine angle had undoubtedly been present since the development of the sinus thrombosis or before.

Comment: This case illustrates a type of complication in which chemotherapy was ineffective. This corresponds to the experience in a number of other cases (i. e., Cases 2, 7 and others unreported) in which the drug was unsuccessful in the presence of a suppurating focus or abscess of considerable size.

DISCUSSION

Diffuse meningitis of otogenic origin has recently been cured by the use of chemotherapy without surgery in many instances, and in several of the cases reported here (Cases 3, 4, 5, 7) the same favorable result might possibly have been obtained without operation.

It has been equally well demonstrated, however, that in the presence of an abscess inside the dura (Case 10) or a focus of necrosis in the temporal bone, either large (Case 2) or of moderate size (Case 8), and in the presence of an extradural abscess (several recent unreported cases), the use of chemotherapy alone fails to sterilize the focus. Concentrations of 14 mg. per cent and over in the blood have been maintained for several days to two weeks without success. Symptoms have usually been relieved but reappeared sometimes before, but usually after, withdrawal of the drug.

Out of the ten cases reported, six (Cases 1, 2, 6, 8, 9, 10) were of this latter type, with large foci of suppuration in which surgical drainage was essential. It appears that chemotherapy alone is most likely to be successful in cases in which a complication has developed rapidly, by extension along vascular channels, before there has been gross destruction of bone, and in the type of bone with pneumatization of the limited small cell type, or in the presence of partial sclerosis. In these types there is less probability of a focus of necrosis or abscess in the bone. However a limited pneumatization does not rule out the possibility of cortical erosion and extra- or subdural ab-

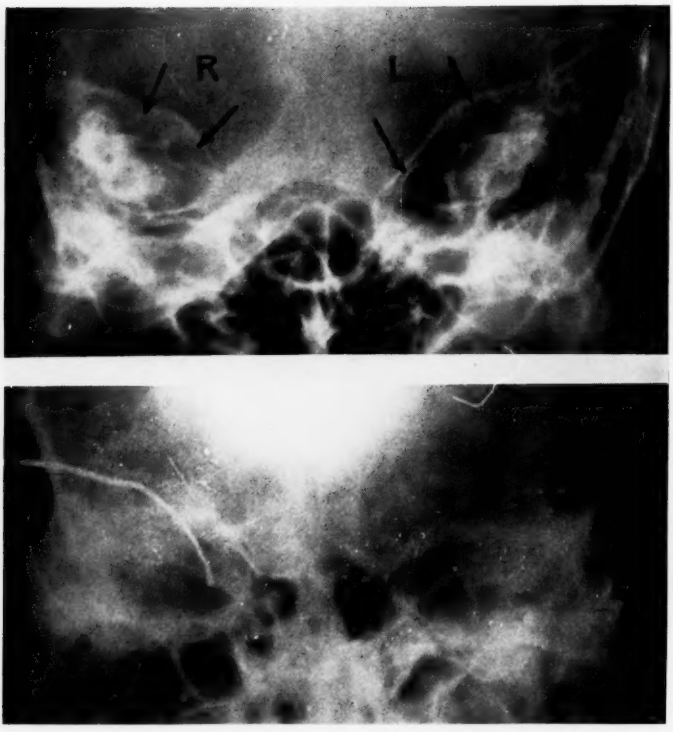

Fig. 1—Case 2. Posterior petrositis. Occipital views of pyramids. The upper view shows extensively pneumatized posteromedial and apex cells bilateral, clouded on the right. The lower view shows the probe lying in the tract made at operation. (In this type the abscess in the apex may be drained by four routes: through the angle, as above; through the subarcuate fossa; by way of the middle cranial fossa; by way of the middle ear and eustachian tube mouth.)

scess. Even cell breakdown cannot always be excluded or diagnosed by X-ray.

Chemotherapy would appear to relieve the urgency of surgery, but in cases where the duration of the suppuration and the clinical evidence indicates suppuration in the mastoid or pyramid, it appears that the surgical exenteration of the cell system along with chemotherapy provides greater safety than reliance on the latter alone.

Because a complication frequently develops suddenly (Cases 8, 9) the protection afforded by chemotherapy should be provided as the initial step in treatment, whenever signs indicate an impending extension of the suppuration.

The masking of symptoms which occurs with chemotherapy apparently can be interpreted as a desirable effect, indicating a definite limitation of the infectious process. It differs from masking of the symptom of pain by sedatives inasmuch as in the former the disease itself is actually impeded. Greater vigilance may be necessary under chemotherapy, but when true surgical indications are present the necessity for surgical drainage is seldom altered by the use of the drug.

Clinical observation appears to indicate that the efficacy of drug therapy depends greatly upon the extent of pneumatization. Suppuration in extensively pneumatized bones tends to resist chemotherapy even in the early stages of the infection (Cases 2, 8) probably because the large air cells permit collection of large amounts of inert infected exudate. In later stages cell breakdown and formation of large necrotic foci are common in this type (Cases 1, 2, 8, 9). Extensive pneumatization is accompanied by development of the air cell system into the pyramid, and consequently the type of case which is likely to develop a necrotic focus in the bone and produce a complication in spite of chemotherapy is the type in which suppuration in the pyramid is a probability.

Other factors which influence the severity of infection and degree of therapeutic success are the virulence of the infecting organism and the degree of resistance on the part of the host as represented by immune bodies in the blood serum.

Blood transfusions have been used, sometimes repeatedly, both in the presence of anemia induced by the infection and for the effect in raising the antibody content. Severe anemia did not occur. The mild anemia which developed in some instances was interpreted as a result of the infection. Evidence of synergistic action of sulfanila-

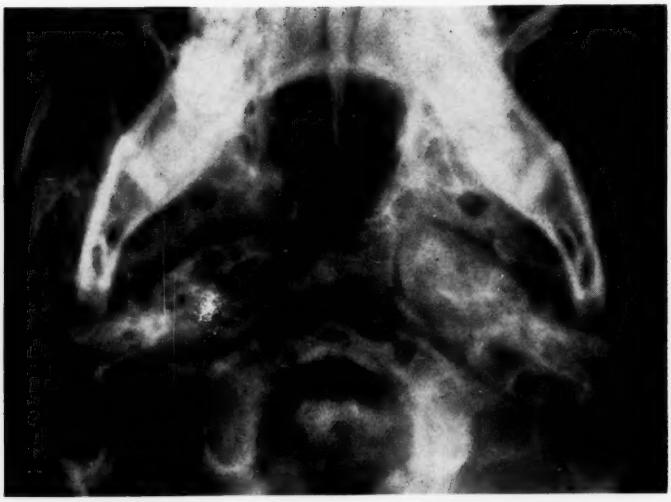

Fig. 2—Case 5. Pneumatized apex bilateral. Clouded on left. Anterior petrositis. Apex cells developed from hypotympanum and therefore were most accessible by way of the middle ear and eustachian tube. Recovery.

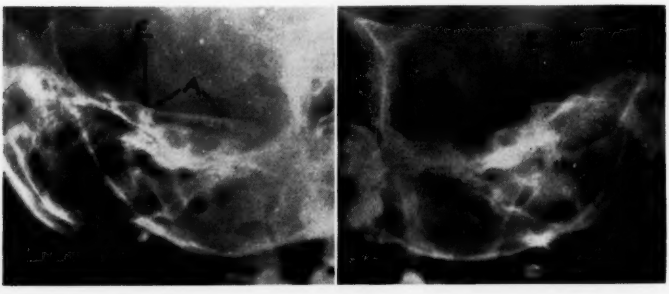

Fig. 3. Stenver's views of pyramids in Case 6. On the right side the mastoid cells are clear, and the superior perilabyrinthine area (A) is pneumatized. On the left side some clouded mastoid cells remain and the superior perilabyrinthine area is pneumatized with two breaks in the cortex (A1). An extradural abscess was drained by elevating dura over the arcuate eminence (E) after mastoid revision. Hem. Strep. meningitis. Recovery.

mide and immune serum has been claimed experimentally and may have clinical significance.

Recurrences or exacerbations of the meningitis have occurred in three cases of this series (4, 5, 7) as well as in several others. In each case the exacerbation occurred after the concentration had been allowed to decrease to around 6 or 7 mgm. per cent when the disease appeared to be subsiding. The disease thereafter has been usually prolonged and more resistant to therapy. It is probable that such exacerbations are due to persistence of suppuration in the cisternae, which becomes diffuse when the concentration of the drug is lowered. Best results have been obtained by obtaining an initial blood level of 15 mgm. per cent and over and maintaining the level for five to seven days or more, decreasing it only two or three days after the spinal fluid has approached the normal.

The treatment for the patient who is desperately ill is not necessarily a criterion of the best policy in the early stages of these infections or in the moderate or mild types of otitic suppuration.

In this series and many others where heavy dosage has been used no serious toxic effects of the drug have been encountered, but there have been great variations in reaction and minor toxic effects have been common. Heavy dosage demands that the patient be under close observation, so that the blood picture, concentrations, urine examination, fluid intake and output may be controlled, which requires hospital care.

The widespread use of these drugs in moderate or light concentration appears to have reduced the number of surgical operations. Recent experience shows however that in certain cases moderate concentrations of the drug do not prevent the development of surgical complications.

SUMMARY

The report includes ten cases. Of these, seven were diffuse suppurative meningitis, one, cisternal meningitis and brain abscess, and two, petrous pyramid suppuration without an intracranial complication. Four of the meningitis cases were secondary to petrositis. All except one were treated in 1939-40, and all were complications of acute middle ear suppuration. There were two fatalities.

(1) The first objective in treatment of the complication is to obtain a blood concentration of the appropriate drug of 15 mgm. per cent or over.

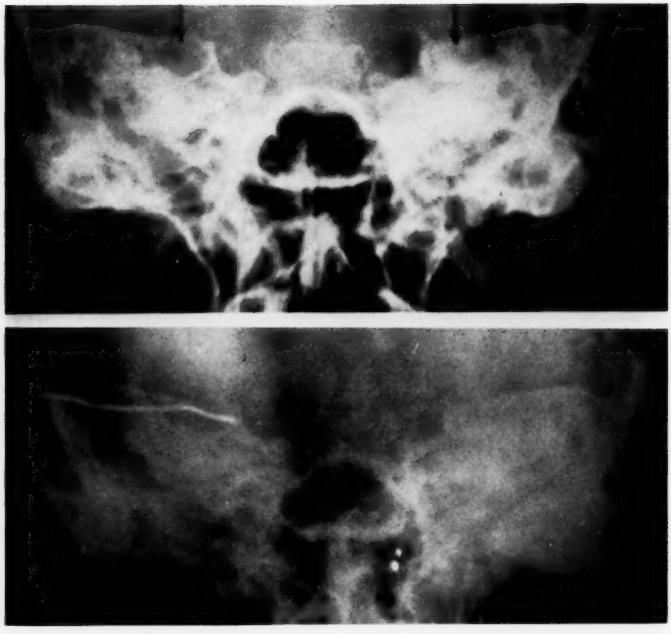

Fig. 4. Occipital views of pyramids in Case 8. A large air cell is seen in the angle of the pyramid on both sides (A) extending medially over the internal meatus. This area is clouded on both sides. The lower view shows the probe lying in the tract made at operation.

(2) When there is evidence of a focus of suppuration in the air cell system or when sufficient time has elapsed for such to form, adequate surgical drainage must still be considered as the next essential; type-specific immune serums, blood transfusions, and supportive measures are next in importance.

(3) The fact that large foci of necrosis or abscesses resist chemotherapy has been demonstrated in several cases.

(4) Premature reduction in the concentration may result in exacerbation and greater resistance to therapy. The blood level should not be reduced until about two days after the spinal fluid has approached normal, since infection may still persist in the cisternae.

(5) Certain cases of ear suppuration resist chemotherapy in moderate concentration even when started at the onset of the disease. The anatomical arrangement constitutes an important factor in the failure of therapy in such cases. Extensive pneumatization of the temporal bone appears to provide conditions in which the drug is least effective, namely, relatively large collections of infected exudate, with tendency to formation of coalescent foci of necrosis.

(6) Six cases of petrositis are reported, five from the past season. Five may be classed as posterior petrositis and one (Case 5) as anterior petrositis. Only the latter case required a radical mastoidectomy.

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XIV

CLOSURE OF PERFORATIONS OF MEMBRANA TYMPANI WITH CARGILE'S MEMBRANE*

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Memphis

An intact ear drum is not essential to good hearing. However it is always to be desired and obtained if possible. The presence of a perforation, even though the middle ear remains dry, is a potential menace. Interference with the projection of hearing, that is, localization of sound, always occurs to some extent, danger of infection is always present, and limitation of sports such as swimming, is also one of the side effects.

The proliferative power of the external or epidermal layer of the membrana tympani, if unimpeded, is tremendous. I am sure we have all seen a drum membrane regenerate, following the radical mastoid operation, until it extends from the margin of the anterior canal wall to the posterior wall of the mastoid, forming a window over the middle ear and the antrum and appearing to be as large as a five cent piece. Traumatic perforations even of great extent are prone to heal spontaneously. Occasionally a perforation, particularly a central one, which has existed for several years, will close spontaneously following an attack of acute otitis media. I believe that an ear drum will always regenerate unless union occurs between the epidermal and mucosal layer, providing the inflammatory process in the middle ear completely subsides.

In a previous paper,[1] I described a phenomenon which I labelled "epidermal escalation" occurring in the membrana tympani and canal wall and set forth these observations on proliferation:

1. The epidermis on the membrana tympani and canal walls does not proliferate and desquamate haphazardly. In the pars tensa, it migrates on its original plane from the anterior margin of the membrane, across the membrane to the wall of the posterior canal and then out along the wall of the canal to the external meatus. This probably explains why a drum which has undergone repeated

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incisions in childhood frequently retains no evidence of scarring in later life.

2. The epidermis of the pars flaccida fans out over the wall of the canal. Thus a dot of india ink is broken down into small particles which move radially from the original locus.

3. The proliferation around the processus brevis is likely to proceed in whorls, but the movement tends to be toward the superior canal wall rather than the posterior.

4. The rate of progress is not uniform but is more rapid in the posterior-inferior quadrant.

5. In no instance have I been able to observe proliferation from the posterior canal wall toward the drum.

The use of a temporary artificial drum membrane to improve hearing was described as early as 1640 by Marcus Banzer. One only has to refer to Politzer's "Diseases of the Ear" to find a complete description of those devised by Toynbee, Hassenstein, and others. Nor is the use of an artificial membrane to promote proliferation of the epidermal layer and thus to close a perforation new, but it has received little attention in the literature and is too frequently ignored.

The most commonly used article is cigarette paper. I have closed perforations with it, but it has several objections, chiefly that it is not resistant to moisture.

Several years ago I found that the ideal material was the mesentery of sheep, marketed by surgical supply houses under the name of Cargile's Membrane, and by all drug stores as fish-skin condom. I prefer the latter because it is thinner and readily available. It is a tough, moisture-proof, pliable membrane, which, probably because it is an animal membrane, is not irritating. I have never noticed any intolerance to it unless one edge is allowed to lap into the tympanum so that it comes in contact with the mucosa. One of its most important qualities is that of adhesion to any surface it comes in contact with, so that once it is applied to the drum membrane, it adheres firmly unless it is floated off. It so nearly resembles the epidermal coat that frequently magnification is necessary to distinguish its outlines. One can observe the margins of the perforation advancing beneath the artificial membrane, and after the perforation has entirely closed the escalatory process begins, so that eventually the membrane is delivered or extruded at the external meatus. I have frequently watched the migration externally until the membrane could be picked up with a forceps without the aid of an ear specu-

lum. It adheres so firmly that in only one instance have I seen it blow off. In the case of a football coach it was necessary to wait until the end of the season, before it could be kept in place. This perforation of two years' duration closed completely with a membrane of sufficient strength to withstand the pressure of two more years of active coaching to my certain knowledge.

The method of application is as follows: The middle ear is carefully inspected for any signs of inflammation, which definitely contraindicates any attempt at closure. If the perforation is of several years' duration, the edges are touched with trichloracetic acid, and one or two days allowed for the resultant inflammation to subside. The canal is carefully cleansed of all cerumen, and if there are any hairs projecting into the canal they must be trimmed to prevent the membrane from adhering to them.

The size of the perforation is estimated. There must be a rim of existent membrane sufficient to support the artificial membrane so it will not sag into the tympanum. The artificial membrane is cut so that it is at least 25% larger than the perforation. A small wisp of cotton is wound on a metal applicator. The tip is moistened and touched to the center of the artificial membrane. This causes it to adhere sufficiently to carry it through the ear speculum and canal provided it does not come in contact with the walls. Application is facilitated if the speculum can be introduced as far as the isthmus of the canal. This straightens the canal, and likewise guards against the membrane's coming in contact with vibrissae or the ceruminous portion of the canal. As soon as the artificial membrane comes in contact with the ear drum it seems to leap into position so that it must be centered exactly over the perforation before it touches. Occasionally, when a small gap is left by reason of the membrane not being exactly centered, it can be teased over this with a well-moistened applicator. In several instances where the perforation has been too large to cover entirely without the center of the artificial membrane sagging into the tympanum, I have reduced the size by first covering only the anterior portion, and then the entire perforation as it becomes smaller.

Healing time varies from ten to thirty days. One can usually tell when the perforation has closed by the change in position of the artificial membrane, that is, by its movement toward the canal wall. I have kept no accurate record of the number of perforations I have closed in the last seven years, but there have been many. I have closed the same aperture twice in one boy at three-year inter-

vals. In both instances the perforation was the result of being kicked in the ear while swimming, the first happening August 8th, 1937, and the second, August 8th, 1940. After the first closure, the regenerated membrane was thin and transparent so that the outline stood out distinctly. For some reason unknown to me, following the second closing the regenerated membrane so closely resembled the uninjured portion that it is difficult to distinguish one portion from the other.

The most spectacular case was the result of a blow from a fist. There was a jagged perforation in the postero-inferior quadrant. Radiating from this was a linear rent extending diagonally up through the short process and another one extending directly anteriorly. The resultant triangular segment was then rolled upon itself until it lay coiled in the antero-superior quadrant. The entire handle of the malleus was bare. I had very little hope of restoring it as I first saw the case 36 hours after the injury, and some thickening had already occurred. The coiled membrane was unrolled, but it persisted in rewinding itself. Under general anesthesia the coiled segment was again ironed out, and a piece of artificial membrane large enough to cover the entire drum membrane, including the perforation, was plastered over it. To my astonishment, the entire membrane healed in thirty days. I saw him about a week ago, ten months after the injury, and there is no evidence even of scarring, and the hearing is normal. Certainly, in this instance, this simple procedure ranked in its value to the patient, by the results obtained, with any surgery in otology.

PHYSICIANS & SURGEONS BLDG.

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XV

FURTHER EXPERIENCES IN FENESTRATION OF THE LABYRINTH IN CHRONIC PROGRESSIVE DEAFNESS: REPORT OF CASES AND AN ANALYSIS OF RESULTS*

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That the apparently permanent improvement of hearing in cases of otosclerosis can be accomplished in many cases by the labyrinth fenestration operation can no longer be disputed by anyone with a knowledge of the progress of this work and a familiarity with the results that have been obtained. Much of the criticism of this surgical treatment has been by persons having an incomplete and inexact knowledge of the principles of the operation, its technic and the details of the results obtained. A fair analysis of such results will undoubtedly show an improvement of hearing of a practical degree which appears to be permanent, in a large percentage of properly selected deaf patients. One basis of criticism, and a proper one, has been the time element in estimating the results. Those cases which have been reported with permanent restoration of practical hearing after an interval of three to six months after operation are open to severe criticism because it is certain that such an interval of time is not sufficient to claim permanency of results. Any statistics of claims for permanent improvement based on any time under a year from operation should be scrutinized with a critical attitude. When the results are analyzed on a basis of a year's elapsed time from operation the percentage of cases with maintenance of improvement is consequently somewhat lowered. Statistical figures are thereby obtained which give a truer and fairer idea of the results which are actually obtained by the operation and should tend to suppress the criticisms based on the time standpoint.

The earliest results obtained through the fenestration operation by the pioneers[1, 2] in this work were almost universally unsuccessful because of early closure of the fistula. The later work of Holmgren[3] and Sourdille[4] however appears to have been at least partially suc-

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cessful, and it is unfortunate that in Sourdille's cases there was not a more exact method of auditory testing because it seems quite definite that many of his patients received much improvement of hearing and have retained open fistulae for periods of six or eight years. Without accurate scientifically recorded measurements of hearing before and after operation his results are generally not acceptable, although it is quite likely they are as good as the results obtained with his modified technic in use at the present time in this country.

The results obtained in the fenestration operation by otologic surgeons in America in the past three years have been impressive as far as the immediate results are concerned. While many of these operated cases have now retained improvement of hearing to a time when the result may be considered permanent there are many others which have lost their early improvement and must be placed in the class of unsuccessful cases, although appearing to be successful during the first few months following operation. The great majority of those otologists performing the operation in this country have followed a surgical technic which is essentially the one advocated and developed by Sourdille[4] but has been modified into a more practical one-stage procedure by Lempert.[5] This technic has given improvement of hearing in over 80 per cent of cases and apparently a permanency of improvement in a sufficiently high percentage of cases to make it a practical and important therapeutic procedure.

One year ago I[6] made a detailed report of the first eight cases operated upon by myself. These cases showed results of a varied character, but in most of them sufficient time had not elapsed to give any certainty of permanent results. Now, after periods of two and a half years to one year and ten months, the results that are present I believe can be considered permanent. As these cases have been reported in some detail[6] the data on them will not be repeated here, but a brief follow-up report on these cases will be made and serial audiograms published of the successful cases. Three of them have retained improvement as follows: One an average gain for conversational frequencies (512, 1024 and 2048) of 31.7 decibels for two and a half years after operation; another, an average improvement of 16.6 decibels for two years after operation; and the third case, an average improvement of 26 decibels for 14 months after operation. In this third case the 26 decibel improvement did not bring the hearing to a practical level because of the severe deafness before operation (77.6 decibel average loss). This patient therefore underwent a revision operation and received further improvement for two or three months but thereafter gradually lost the improve-

ment, and at the present time (seven months after the revision) the hearing is slightly worse than before the revision.

The remaining five cases of this group were unsuccessful. Two showed prompt closure of the fistula, and on revision both again resulted in closure of the fistula with an average loss of 22 decibels two years after operation in one case and an average loss of 2 decibels two years after operation in the other case. In two of the eight cases the hearing has been made worse. Both were unsuitable for the operation, since the bone conduction was decreased and one patient had a dead labyrinth. The remaining case was one in which the external semicircular canal was fistulized in a mastoid that had been radically operated on some years before. This patient showed no improvement at any time after operation and the result after two years was 12.7 average decibel loss.

At this time I shall make a report in some detail of twelve additional cases which have been operated upon for more than one year. Such an elapsed time should preclude any further change in the fistula as far as reparative processes are concerned. These patients were operated upon according to the principles of Sourdille[4] through the endaural technic as outlined by Lempert[5] except for some modifications in certain cases. The audiograms were done with extreme care largely without reference to previous audiograms and using a masker in the untested ear. In expressing any change of hearing in the following reports an average reading of the three frequencies, 512, 1024 and 2048, is used. For instance a 15 decibel gain of hearing means an average of 15 decibels for these three frequencies.

REPORT OF CASES

CASE 9.—(M. R.), a woman 38 years of age, first noticed deafness after the birth of a child 15 years ago. Since then there had been gradually increasing deafness of both ears, especially the left, with intermittent tinnitus worse in the left ear. During the past two years there had been rapid progression of the deafness. There was no history of ear abscesses. The tonsils and adenoids had been removed in childhood, and fourteen years ago bone had been removed from the nose and adhesions broken up in the throat. An older sister was somewhat deaf, but otherwise there was no history of familial deafness. Considerable local treatment during the past few years had given no improvement. Examination of the ears showed both tympanic membranes thickened with two healed perforations in the left. Tuning fork tests showed considerable impairment of hearing in the left ear and moderate impairment in the right, particularly to low tones, with normal bone conduction and no lateralization of tone. Operation was performed on the left ear on March 29, 1939, but the head of the malleus was not removed as the malleus and incus could not be separated due apparently to bony ankylosis of the joint. There was moderate dizziness on fistulizing the external semicircular canal

and considerable immediate improvement of hearing to voice tests. About five weeks after operation the patient developed a middle ear abscess in the operated ear which ruptured through the drum and drained pus for five weeks. During the course of this abscess there was no increased dizziness and no general symptoms. The operative wound healed completely and the perforation accompanying the abscess closed, but the hearing gradually became worse until it was 11 decibels below the preoperative level. The fistula has remained moderatively active.

Comment: One year and eight months after operation there is a 13.3 decibel loss of hearing over the preoperative level. Four weeks after operation the hearing had improved 23.4 decibels but subsequently was gradually lost. Six factors may have contributed to the poor result: (1) The head of the malleus was not removed. (2) The tympano-meatal flap was torn and it was necessary to dissect one from the anterior superior canal wall to cover the fistula. (3) A middle ear abscess developed in the operated ear five weeks after operation. (4) There has been some closure of the fistula as indicated by a lessened fistula reaction from its optimum. (5) The considerable loss of hearing in the unoperated ear may indicate a rapid progression of the basic cause of the deafness. (6) The best prerequisites for the operation were not present as the scarred drum with healed perforations indicated middle ear abscesses at some previous time.

CASE 10.—(E. G.), a woman 33 years of age, gave a history of increasing deafness of both ears for about five years with loud tinnitus in both. There was no history of ear abscesses, no dizziness and no chronic nose or throat troubles. Much treatment of the nose and ears had given no improvement. Examinations showed no abnormality of the nose or nasopharynx, and the ears were negative except for slight thickness of the drums. . Tuning fork tests gave considerable impairment of hearing, slightly more marked in the right ear, with normal bone conduction in both and lateralization of tone to the right ear. A series of inflation treatments for a month gave no improvement of the hearing or tinnitus. Operation was performed on the right ear on May 5, 1939. The malleus head was not removed. The best hearing improvement was obtained three weeks after operation when there was an average gain of 20 decibels. Following this there was a gradual loss of the improvement. At the end of four months from operation the hearing had returned to its preoperative level where it remains. A revision operation was performed on September 19, 1940, 16½ months after the original operation. The fistula was found filled partially by regenerated bone but mostly by fibrous tissue. Following the revision no positive fistula test could be elicited, but one month later the hearing had improved 13 decibels. A few weeks later, however, the hearing had returned to its pre-revision level.

Comment: The factors concerned in the failure in this case may be the incompleteness of the operation (malleus head was not removed) or may be the closing of the fistula. The absence of a positive fistula test following the revision in spite of an improvement of the hearing is an interesting feature which appears difficult of explanation.

CASE 11.—(E. S.), a girl 23 years of age, had noted deafness of the right ear for about 12 years and of the left ear for a shorter time, with intermittent rumbling noises in both, particularly in the right ear. She had had several attacks of suppurative otitis media in the right ear. There was no history of deafness in her father or mother. The tonsils had been removed four years before and also

bone removed from her nose. In spite of treatment the deafness had been getting worse in the last few years. Examination revealed no pathology in the nose or nasopharynx, but both tympanic membranes were thickened and scarred with a moderate-sized perforation in the lower posterior quadrant of the right drum and a large perforation in the posterior part of the left drum. Tuning fork tests showed considerable impairment in both ears, more in the right, with normal bone conduction and no lateralization of tone. On inflation of the eustachian tubes air appeared to enter both middle ears easily and well. Operation was performed on the right ear on May 12, 1939. One month after operation the hearing had improved an average of 26.6 decibels. Thereafter some of the hearing improvement was lost, but 18 months after operation a hearing gain of 15 decibels had been retained with a moderately active fistula.

Comment: It is of interest to note that there has been maintained a moderate improvement of hearing in spite of the history of middle ear abscess and a scarred and perforated drum. The best improvement was not retained, apparently because of partial closure of the fistula. During the interval since operation this patient became pregnant and delivered without any effect on the hearing.

CASE 12.—(A. B.), a man 22 years of age, had had a severe deafness of the left ear with loud buzzing tinnitus since cerebrospinal meningitis ten years ago. The deafness had apparently not been increasing. He had noted no deafness of the right ear. He complained of frequent mild dizzy spells, not accompanied by nausea. Tonsils had been removed. Examination showed tympanic membranes normal in appearance and normal eustachian orifices. Tuning fork tests showed profound deafness of the left ear and very slight deafness of the right ear with only fair bone conduction in both and lateralization of tone to the right ear. Operation was performed on the left ear on May 19, 1939. On fistulizing the external semicircular canal no dizziness was noticeable and no improvement of hearing. However the patient remarked that the tinnitus had ceased. Three weeks after operation an audiogram showed no change whatsoever in the hearing of the operated ear. Sixteen months following operation, however, audiograms showed an average of 21 decibel improvement. The fistula remains very active, and the tinnitus has not returned except occasionally for only a few seconds at a time.

Comment: It was realized with this patient that the chances for improvement of the hearing were slight because of the history of the deafness following meningitis and because of only fair bone conduction. The operation was performed solely in an attempt to improve the tinnitus which was extremely distressing to the patient. To date the operation has been entirely successful from that standpoint. There has been no hearing improvement of benefit to the patient.

CASE 13.—(W. L.), a man 43 years of age, had noted deafness of both ears since childhood. For the past five years it had been getting decidely worse, particularly in the right ear. He did not complain of tinnitus or vertigo. There was no history of ear abscesses and no deafness in his family. Examination showed the nose and eustachian orifices normal and the tympanic membranes negative, except for slight thickening. Tuning fork tests showed profound deafness of the right ear and considerable of the left with probably normal bone conduction and doubtful lateralization of tone. Operation was performed on the right ear on June 4, 1939. On fistulizing the external semicircular canal only slight dizziness was noted and probably no improvement of hearing. There was no improvement of

hearing during the first three months after operation, but 15 months after operation audiograms show an average gain of 21.7 decibels in the operated ear.

Comment: The absence of hearing improvement and a positive fistula test during the three months following operation was probably due to a serous labyrinthitis. This patient did not report for examination during the period of a year and audiograms then, 15 months after operation, showed a gain of 21 decibels with a fairly active fistula. This hearing gain has been of no practical importance to the patient because it has not been improved to a practical level due to the profound deafness before operation.

CASE 14.—(M. H.), a woman 27 years of age, had been very deaf in both ears, worse in the left, for twelve years. Deafness came on suddenly at 15 years of age when she was in a house struck by lightning. She was unconscious for a short time, and on recovery she had developed a left-sided hemiplegia, facial paralysis and extreme deafness. The hearing improved to a slight extent during the few months following this accident, and she entirely recovered from the hemiplegia. During the past ten years, however, the hearing has gradually became worse with roaring noises in both ears. There was no history of ear abscesses and no deafness in the family. Considerable treatment of the ears had given no improvement. Tuning fork tests showed considerable deafness of both ears, worse in the left, with probably less than normal bone conduction and lateralization of tone to the left ear. Operation was performed on the left ear on June 21, 1939. The malleus head was not removed. The hearing became worse after operation and has not improved since. Fifteen months after operation there is a 12 decibel loss of hearing over the preoperative level, and the fistula is very active.

Comment: In spite of a very active fistula there has been a loss of hearing in this patient. We must assume that the deafness was of the perceptive type and the patient therefore unsuitable for the operation. It was very difficult to estimate the bone conduction because of poor cooperation of the patient.

CASE 15.—(R. J.), a man 27 years of age, gave a history of gradually increasing deafness of both ears for six years with buzzing tinnitus which at times was severe. He did not complain of vertigo and had no symptoms referable to the nose and throat. His father's hearing was somewhat impaired, but there was no deafness in his mother, six brothers or three sisters. There was no history of ear abscesses. He had had a great variety of treatment to his nose and ears without any improvement. Examination showed the nose and nasopharynx negative and normal appearing tympanic membranes. Tuning fork tests showed considerable impairment of hearing in both ears, particularly to low tones, with normal bone conduction in both and slight lateralization of tone to the left ear. Operation was performed on the left ear on June 23, 1939. On fistulizing the external semicircular canal there was moderate dizziness and marked improvement of hearing to voice tests. The malleus head was not removed, and the tympano-meatal flap was torn, necessitating the cutting of a skin flap from the anterior-superior canal wall to place over the fistula. Following the operation there was only slight improvement of hearing at any time and the end result (16 months after operation) was a few decibels of improvement with only a slightly active fistula.

Comment: The negative result in this case has been attributed to errors in the technic. The bone conduction was undoubtedly good and the tympanic mem-

brane and vestibular function normal. The failure to remove the malleus head and the laceration of the tympano-meatal flap probably caused the failure. At a convenient time this patient will have a revision operation at which time the head of the malleus will be removed.

CASE 16.—(F. B.), a man 44 years of age, had had gradually increasing deafness of both ears, especially the left, for about 12 years, with buzzing noises in both but louder in the left. He never had ear abscesses but was continually getting nasal and sinus infections. He had had a submucous resection and tonsillectomy several years previously and had received much nasal and inflation treatments by various physicians without improvement of his hearing. Examination showed the tympanic membranes somewhat thickened and retracted, but no scars or perforations. Tuning fork tests showed considerable deafness of both ears, more marked in the left to all tones, with normal bone conduction and lateralization to the left ear. Operation was performed on June 27, 1939 on the left ear. The malleus head was not removed and the tympano-meatal flap was torn, necessitating the cutting of a skin flap from the anterior-superior canal wall to cover the fistula. The best hearing improvement was obtained one month after operation when there was an average gain of 25 decibels over the preoperative hearing. Thereafter this improvement was gradually lost with a gradually lessening fistula reaction. Fourteen months after operation there was a 6.6 decibel loss of hearing over the preoperative level with only a slightly active fistula.

Comment: This case has been a failure largely because of the gradual closure of the fistula. Perhaps the errors in the technic contributed also, but it seems more likely that the good improvement present one month after operation was not maintained because of partial closure of the fistula.

CASE 17.—(J. W.), a man 26 years of age, first noticed buzzing noises intermittently in right ear four years ago and some deafness three years ago. Since then the deafness and tinnitus have been gradually getting worse in the right ear, and for the past two years the left ear has been getting deaf. There is no history of ear abscesses or deafness in family. Examination showed both ear drums somewhat thickened, but otherwise negative. There was no pathology in the nose, and the tonsils had been removed. Tuning fork tests showed considerable deafness in both ears, slightly more in the left, with prolonged bone conduction which was better in the right ear, with lateralization to the right. Operation was performed on the left ear on September 8, 1939, with no errors in the technic. The best hearing improvement was present 20 days after operation when an average gain of 36.4 decibels was registered. During the following six months there was a gradual loss of most of this improvement co-incident with a lessening response of the fistula. Seven months after operation there was only an average improvement of 8 decibels, and at the present time, 16 months after operation, there are still 8 decibels of improvement with only a slightly active fistula present.

Comment: The early improvement in this patient's hearing was extremely gratifying but gradual closure of the fistula resulted in a loss of practically all improvement, and the end result has been of little or no benefit to the patient. This patient will have a revision at a favorable opportunity.

CASE 18.—(A. Z.), a woman 38 years of age, gave a history of gradually increasing deafness of both ears, especially in the left, for five years. There was no

tinnitus present, no dizziness and no history of ear abscesses. Her mother became deaf in middle age. There were no symptoms referable to the nose and throat. Examination showed both ear drums somewhat thickened and retracted with probable scars on the left. Tuning fork tests showed moderate deafness in the right ear and considerable in the left, particularly to low tones, with prolonged bone conduction and lateralization to the left ear. Operation was performed on the left ear on September 15, 1939, with no errors in the technic. Six weeks after operation the hearing had improved an average of 36.7 decibels over the preoperative level. Thereafter there was a gradual loss of some of this improvement with a lessening fistula response. Seven months after operation there was only 18.3 decibels improvement and at present, 15 months after operation, there has been retained a gain of 17 decibels over the preoperative hearing with only a slightly active fistula present.

Comment: This patient is another example of excellent immediate improvement, most of which has been subsequently lost by closure or partial closure of the fistula. In these last two cases the endosteum was not removed when the external semicircular canal was fenestrated. The incomplete opening of the canal very probably contributed to its closure after operation.

CASE 19.—(J. K.), a man 50 years of age, had had some deafness for years, but for the past four months it had increased considerably in both ears. At times the hearing was extremely bad, and there were intermittent pounding noises in both. There was no dizziness, no history of deafness in the family and no ear abscesses at any time. Considerable treatment of various kinds in the past two years had given no improvement. Examination showed ear drums negative and no pathology in nose or throat. Tuning fork tests showed considerable deafness in the right ear and moderate in the left with probably normal bone conduction in the left ear but somewhat reduced in the right. Operation was performed on the right ear on October 20, 1939, with no errors in the technic. There was no improvement in the hearing at any time following operation and only a slightly active fistula was present. At the present time, 13 months after operation, there is 23.4 decibel loss of hearing over the preoperative level and a negative fistula test.

Comment: This patient was not suitable for operation. His bone conduction was reduced from normal too far to obtain a favorable result. The slightly active fistula may also have indicated a weakened vestibular function.

CASE 20.—(N. H.), a woman 40 years of age, gave a history of gradually increasing deafness of both ears for eight or nine years with buzzing and ringing noises in both constantly. There was no dizziness. She had had several attacks of suppurative otitis media in both ears but none in the right one for several year. There was no deafness in three brothers and one sister. Examination showed both ear drums much distorted with scars, thickening, adhesions and perforations. There was no evidence of infection. The nose showed fairly advanced atrophic rhinitis. Tuning fork tests showed considerable deafness of both ears with prolonged bone conduction in both and lateralization to the right ear. Operation was performed on the right ear on November 10, 1939, with no errors in the technic. Subsequent to the operation there was a loss of hearing in spite of a very active fistula. There was no improvement at any time, and the end result, one year after operation, was a 3.3 decibel loss over the preoperative level, with a very active fistula response.

Comment: This patient possessed the proper degree of deafness, normal bone conduction, normal vestibular function and was operated upon without error of technic and with the creation of a very active fistula. In spite of these favorable factors there was no improvement of hearing at any time. The cause of failure must be attributed to changes in the drum and middle ear which had occurred as a result of recurrent attacks of suppurative otitis media. It appears probable that the function of the round window had been lost because of fibrotic and adhesive changes.

SUMMARY OF RESULTS IN THIS SERIES OF TWELVE CASES

An analysis of these twelve cases will show results of a variable character. On the whole the results are not good. This is largely due to operation of patients who did not have the proper prerequisites and partly perhaps to the fact that the malleus head was not removed in five cases. Some of these cases were operated upon in spite of imperfect prerequisites in an endeavor to ascertain whether or not improvement of hearing could be obtained in patients who had had middle ear infections at one time. Others were operated upon with a modified technic (without removal of the malleus head) in order to ascertain if the hearing could be improved and maintained without the necessity of going through the time-consuming procedure of removing the head of the malleus.

Six of these 12 operated patients have obtained improvement of hearing for more than one year, while the other half have lost some hearing. The improvement in two of the six cases has been good, in two cases, has been moderate, and in two, has been slight. In the two cases that received the most improvement of hearing, 21.6 and 21.7 decibels respectively, as registered by audiograms, there has been no subjective improvement because of profound deafness before operation. In one of these, however, the tinnitus present before operation has disappeared, and the patient is extremely well pleased.

The two patients who received a moderate improvement of hearing, 13 and 17 decibels respectively, have subjectively been much benefited. Their preoperative hearing was of a degree where this slight gain has brought the hearing to a practical level. At the same time the lessening of the tinnitus has been helpful.

The two patients that received improvement of 6.7 and 8.3 decibels respectively have noticed some change for the better. One of these patients showed considerable gain of hearing shortly after operation with the subsequent loss of most of this improvement.

Of the six cases in this group which were failures, three had good bone conduction, although two of them had scarred drums.

All three received excellent improvement of hearing shortly after the operation, but subsequently lost all the improvement apparently because of closure of the fistula with perhaps some errors in the technic contributing to it. Two of the unsuccessful cases had poor bone conduction and received no improvement at any time after operation. One patient had the proper indications for operation except a scarred and perforated drum. This one received no improvement at any time.

Let us consider these twelve cases briefly from the standpoint of operative indications. Five of them (Cases 10, 12, 13, 15 and 17) possessed the essential indications for operation, that is conductive deafness, normal or only slightly reduced bone conduction, normal vestibular function and normal tympanic membranes. Two of these, however, had such profound deafness that improvement of hearing to a practical level could not have been expected. Of the seven patients who did not possess the proper indications (Cases 9, 11, 14, 16, 18, 19 and 20), five showed evidences of middle ear abscesses at some previous time, three (Cases 9, 16 and 18) showed scarred drums with healed perforations, and two (Cases 11 and 20), showed scarred drums with actual perforations present. The remaining two patients with improper indications showed bone conduction reduced below a favorable level.

Of the five patients who possessed the favorable operative indications four retained improvement of hearing beyond the one year period. The hearing gain was respectively 21.6, 21.7, 6.7 and 8.3 decibels. The fifth case with proper operative indications lost an average of 1.7 decibels after 16 months.

Of the seven patients who did not possess the proper operative indications two retained improvement of hearing as follows: one gained 15 decibels at the end of 18 months and the other gained 17 decibels at the end of 15 months. The remaining five patients with unfavorable operative indications lost some hearing over the preoperative level.

Of this series of 12 operated patients nine received satisfactory improvement of hearing shortly after operation. Only two of these, however, retained this greatest improvement. Of the remaining seven patients, four lost some of the early improvement, but have retained some gain over the preoperative level, while three patients have not only lost all the early improvement but the end result is a slight loss over the preoperative level.

Three patients received no improvement of hearing at any time after operation. Two of these had poor bone conduction before operation, and the other one had a much scarred, thickened and retracted drum containing a large perforation.

Five of these 12 operated patients had tympanic membranes which showed evidences of middle ear abscesses at some previous time. Three of them showed healed perforations, and two had actual perforations in the drums. One of the patients with a perforation in the drum has retained a hearing gain of 15 decibels, while the other one with a perforation has lost 3.3 decibels. The three patients with healed perforations of the drums showed hearing changes as follows: 13.3 decibel loss, 6.6 decibel loss and 17 decibel gain. Of the five patients, therefore, with scarred and perforated drums, two received a gain of hearing of 15 and 17 decibels respectively, while the remaining three lost some hearing over the preoperative level.

Five of this series of twelve patients were operated upon with a modified technic in that the head of the malleus was not removed (Cases 9, 10, 14, 15 and 16). Four of these received very satisfactory improvement of hearing shortly after operation. During the subsequent few months, however, all of the improvement was lost in three cases, and the fourth case retained only 6.7 decibel gain 15 months after operation. One of the five cases in which the malleus head was not removed received no improvement at any time, and the end result was 11.7 decibel loss over the preoperative level. This patient showed poor bone conduction before operation.

In these 12 operated patients the fistula reaction has remained positive in 11 although varying somewhat in the degree of reaction. I have divided the fistula reaction into four grades as follows: the negative reaction, the slightly positive, the moderately positive and the strongly positive reactions. Two of the 12 patients have retained a strongly positive fistula reaction, although both of them have lost some hearing over the preoperative level as the end result. Neither of these patients received improvement of hearing at any time after operation. Three patients retained a moderately positive fistula reaction. Two of these gained 15 and 21.6 decibel improvement respectively as the end result, while the third case lost 13.3 decibels. Six patients have retained a slightly active fistula, four of which have retained 21.7, 6.7, 8.3 and 17 decibel improvement respectively. The other two patients lost 1.7 and 6.6 decibels over the preoperative level.

In 11 of the 12 cases the fistula reaction was strongly positive shortly after operation but was subsequently diminished in all but two cases. The lessening fistula reaction was co-incident with a gradual loss of some of the hearing improvement in most of the cases. As a rule the hearing improvement paralleled closely the fistula reaction. As the fistula reaction gradually diminished the improvement was gradually lost. However in some cases the hearing diminished to below the preoperative level, while a slightly positive fistula reaction still remained.

In the two cases in which the fistula reaction did not diminish from its most active state there was no improvement of hearing at any time after operation. Both of these patients did not have favorable operative indications, one having poor bone conduction and the other having a much scarred, thickened and perforated drum. One patient who had poor bone conduction before operation showed a negative fistula reaction at all times postoperatively co-incident with a reduction of hearing.

Postoperative complications were observed in two cases. Six weeks after operation a suppurative otitis media developed in the operated ear of one patient with rupture of the tympanic membrane. Purulent discharge persisted for about one month. During this time there was no increased dizziness and no general symptoms. In this patient the end result has been a 13 decibel loss of hearing over the preoperative level, but the fistula reaction has remained moderately strong. The other complication was a partial facial paralysis which started to develop the day following the operation. The paralysis did not become complete and began to clear up ten days after operation. The facial muscular action was completely restored one month after operation.

In analyzing the results in this series of 12 cases it is only fair that consideration be given to the fact that what is now known as the proper operative indications were not present in the majority of them. It has been only by the operation of such cases that it has been determined just what patients are suitable and what patients are not suitable for the operation. In a small number of operated cases, such as is herewith reported, it would hardly be justifiable to make any positive assertions on operative indications, but at least certain impressions can be gained on one or two features concerning the indications and technic of the operation.

In studying the audiometric records of those five cases (Cases 9, 11, 16, 18 and 20) whose histories and examinations indicated that

the operated ears had been involved in one or more attacks of suppurative otitis media at some previous time, it is noteworthy to observe that four of them gave very satisfactory improvement of hearing shortly after operation, and two of these four retained a hearing improvement of 15 and 17 decibels respectively after a one year period. Whether the deafness in these cases was due to changes set up in the middle ear by the former suppurative process or whether it was due to a co-incident otosclerosis was not ascertainable. The fact remains, however, that some cases of this kind can be improved by operation, and it is my impression that the reason for the failure of two of these five cases to retain improvement of hearing was gradual closure of the labyrinthine fistula rather than any defects present in the middle ear. The reason that one case of these five received no improvement of hearing at any time after operation (Case 20) appears to be that the changes in the middle ear secondary to the frequent attacks of suppurative otitis media had disturbed the function of the round window. From the observation of these five cases, therefore, the impression can be gained that at least some of the cases giving history of middle ear abscesses can be satisfactorily improved by the fenestration operation. It would seem desirable to establish, if it is possible to do so, the condition of the round window and its function before operation in such cases and thereby determine whether or not such a case is suitable for the operation from that standpoint.

Further impressions can be gained from the observation of those five patients (Cases 9, 10, 14, 15 and 16) in which the malleus head was not removed. Four of these received satisfactory improvement of hearing shortly after the operation, as follows: Case 9 gained 23.4 decibels four weeks after operation; Case 10 gained 20 decibels three weeks after operation; Case 15 gained 10 decibels one month after operation; and Case 16 gained 25 decibels one month after operation. The remaining patient (Case 14) received no improvement at any time after operation which was attributed to the fact that her bone conduction was not good. Of these four patients that received improvement shortly after operation, two (Cases 9 and 16) were in the group of those who had had suppurative otitis media at some previous time.

Of the four cases who received early improvement in this group in which the malleus head was not removed, only one retained improved hearing beyond the one year period (Case 15), and this improvement was only an average of 6.7 decibels. It may be argued from the negative end-result in these five cases that the reason for

the failure was the fact that the malleus head was not removed, thereby preventing Shrapnell's membrane from being placed over the fistula and thus setting up conditions conducive to closure of the fistula with the resultant loss of the early improved hearing. Such a contention is not logical for two reasons. In the first place it seems assured that in the great majority of cases, and perhaps in all of them, the drum is not displaced sufficiently to allow of Shrapnell's membrane being placed over the fistula. Secondly, it was observed that in two of these five patients mentioned above (Cases 9 and 14) the fistula reaction has remained strongly positive in one instance and moderately positive in the other. This should indicate that there had been little or no closure of the fistula. Why then was no improvement retained in these two cases? In one instance (Case 14) there was no improvement at any time apparently because of perceptive type of deafness (poor bone conduction). In the other instance (Case 9) a middle ear abscess developed five weeks after operation and may have been the cause of the subsequent loss of hearing.

In three of the five patients in which the malleus head was not removed (Cases 10, 15 and 16), the fistula has remained only slightly active after one year. Two of these (Cases 10 and 16) have lost all the early improvement, and the other one (Case 15) has retained 6.7 decibels improvement.

From a study of these five cases the impression is gained that the negative end results are due to the nearly complete closure of the fistula in three cases and to the ear abscess and perceptive deafness in the other two cases, rather than to the fact that the malleus head was not removed. Whether the failure to remove the malleus head contributed to the closure of the fistula in three of these cases is uncertain. No general deduction or conclusion is justifiable from observation of such a small number of cases. It would seem desirable to operate upon more patients by this modified technic before reaching any conclusion regarding it.

CONCLUSION

In the above report of this series of 12 cases it has been the intention to present them not with the idea of emphasizing good results that may be obtained by the fenestration operation, but rather with the thought of drawing attention to such features as errors of technic, results with modified technic, results to be observed and expected with certain anatomic features that ordinarily are thought to make the case unsuitable for operation and to present data that will permit analysis from various standpoints. In the near future

I shall make a further report of 20 additional cases which have been selected with more discrimination as to the operative indications and which show every evidence at present of an extremely satisfactory end result.

SUMMARY

Detailed reports with serial audiograms are herewith presented of twelve patients who received the labyrinth fenestration operation for chronic progressive deafness.

In analyzing the results in these patients after an elapsed time from operation of a year or more the following information and impressions have been gained.

1. Six patients have obtained improvement of hearing and six patients have lost some hearing as the end result.

2. This generally poor result has been attributed largely to improper selection of patients in the majority of the cases.

3. Five of these operated cases possessed the proper operative indications and four of them retained improvement of hearing.

4. Of the seven patients who did not have favorable operative indications, two retained improvement of hearing.

5. Five patients of this series showed evidences in the tympanic membranes of having had suppurative otitis media at some previous time. Two of this group retained improvement of hearing.

6. In five of the twelve operated cases, the malleus head was not removed. One of this group retained a slight improvement of hearing.

7. The results indicate that at least some of the cases in which there have been middle ear abscesses at some previous time can be improved by the operation.

8. There is some reason to believe that failure to remove the malleus head may not contribute to closure of the labyrinthine fistula.

2035 DELANCEY STREET.

NOTE.—The appended tables have been abridged to accommodate the available space. Those in the original manuscript contained many more notations for which the reader may refer directly to the author.—EDITOR.

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CASE 1. AUDIOMETRIC RECORDINGS BEFORE AND AFTER OPERATION ON RIGHT EAR JUNE 29, 1938

	Right Ear									Left Ear								
	Audiometer Frequencies								Aver.	Audiometer Frequencies								Aver.
Dates	128	256	512	1024	2048	4096	5792	8192	Dec. Loss	128	256	512	1024	2048	4096	5792	8192	Dec. Loss
6-18-38	58	59	67	53	85	88	100	100	68.3	42	43	55	38	67	82	100	100	53.3
9-16-40	30	45	35	25	50	85	100	100	36.6	50	45	40	40	80	80	100	100	53.3

CASE 6. AUDIOMETRIC RECORDINGS BEFORE AND AFTER OPERATION ON RIGHT EAR ON OCT. 12, 1938

	Right Ear									Left Ear								
	Audiometer Frequencies								Aver.	Audiometer Frequencies								Aver.
Dates	128	256	512	1024	2048	4096	5792	8192	Dec. Loss	128	256	512	1024	2048	4096	5792	8192	Dec. Loss
9-26-38	65	70	85	80	65	77	100	100	76.6	77	73	76	67	39	67	68	100	60.6
10-22-38	58	48	47	37	35	72	100	100	39.6	68	69	82	70	45	75	65	100	65.6
9-16-40	65	75	75	60	45	70	100	100	60.	100	85	80	80	50	70	100	100	70

CASE 8. AUDIOMETRIC RECORDINGS BEFORE AND AFTER OPERATION ON RIGHT EAR ON JAN. 11, 1939
REVISION OPERATION ON APRIL 2, 1940

	Right Ear									Left Ear								
	Audiometer Frequencies								Aver.	Audiometer Frequencies								Aver.
Dates	128	256	512	1024	2048	4096	5792	8192	Dec. Loss	128	256	512	1024	2048	4096	5792	8192	Dec. Loss
1- 5-39	56	55	74	68	90	100	100	100	77.6	26	33	41	46	63	78	100	100	50.
2-23-39	15	20	32	25	42	85	100	100	33.	43	40	37	48	65	79	100	100	50.
3-27-40	50	50	60	45	50	100	100	100	51.6	40	35	40	50	70	85	100	100	53.3
5-16-40	20	20	40	25	50	80	100	100	38.3	30	35	40	50	70	85	100	100	53.3
9-23-40	30	35	45	50	75	90	100	100	56.6	50	55	45	50	70	100	100	100	55.

CASE 9. AUDIOMETRIC RECORDINGS BEFORE AND AFTER OPERATION ON LEFT EAR MARCH 29, 1939

	Right Ear									Left Ear								
	Audiometer Frequencies								Aver.	Audiometer Frequencies								Aver.
Dates	128	256	512	1024	2048	4096	5792	8192	Dec. Loss	128	256	512	1024	2048	4096	5792	8192	Dec. Loss
3-27-39	40	50	45	45	25	35	45	50	38.3	65	65	55	50	45	65	55	60	50
4-24-39	55	50	45	45	25	35	50	60	38.3	50	45	45	20	15	35	60	55	26.6
10-30-40	100	75	70	70	35	45	65	50	58.3	100	75	70	65	50	55	55	30	61.6

CASE 10. AUDIOMETRIC RECORDINGS BEFORE AND AFTER OPERATION ON RIGHT EAR ON MAY 5, 1939
REVISION OPERATION ON SEPT. 19, 1940

	Right Ear									Left Ear								
	Audiometer Frequencies								Aver.	Audiometer Frequencies								Aver.
Dates	128	256	512	1024	2048	4096	5792	8192	Dec. Loss	128	256	512	1024	2048	4096	5792	8192	Dec. Loss
5- 2-39	60	50	45	40	25	35	35	25	36.6	35	35	35	30	10	35	20	35	25.
5-26-39	35	20	15	15	20	50	55	50	16.6	45	35	45	40	25	50	15	35	36.6
8- 1-39	55	45	40	45	15	45	45	40	33.3	30	30	35	20	25	45	20	20	26.6
10- 7-40	60	55	45	30	20	80	100	100	31.6	30	30	45	35	20	50	30	25	33.3
10-31-40	65	55	60	30	20	70	55	100	36.6	35	40	45	35	20	45	30	35	33.3

CASE 11. AUDIOMETRIC RECORDINGS BEFORE AND AFTER OPERATION ON THE RIGHT EAR ON MAY 12, 1939

	Right Ear									Left Ear								
	Audiometer Frequencies								Aver.	Audiometer Frequencies								Aver.
Dates	128	256	512	1024	2048	4096	5792	8192	Dec. Loss	128	256	512	1024	2048	4096	5792	8192	Dec. Loss
5-10-39	60	55	50	40	35	50	45	25	41.6	45	25	25	35	15	35	30	20	25.
6-13-39	20	25	30	5	10	30	30	20	15.	35	25	35	40	20	30	40	35	31.6
8-14-40	30	40	35	20	25	35	30	30	26.6	25	30	30	30	10	15	10	30	23.3

CASE 12. AUDIOMETRIC RECORDINGS BEFORE AND AFTER OPERATION ON LEFT EAR ON MAY 19, 1939

	Right Ear									Left Ear								
	Audiometer Frequencies								Aver.	Audiometer Frequencies								Aver.
Dates	128	256	512	1024	2048	4096	5792	8192	Dec. Loss	128	256	512	1024	2048	4096	5792	8192	Dec. Loss
5-13-39	20	10	10	15	5	0	30	5	10.	65	65	95	100	95	75	100	100	96.6
9-17-40	20	15	15	10	5	5	20	15	10.	100	75	75	70	80	70	70	50	75.

CASE 13. AUDIOMETRIC RECORDINGS BEFORE AND AFTER OPERATION ON RIGHT EAR ON JUNE 14, 1939

	Right Ear									Left Ear								
	Audiometer Frequencies								Aver.	Audiometer Frequencies								Aver.
Dates	128	256	512	1024	2048	4096	5792	8192	Dec. Loss	128	256	512	1024	2048	4096	5792	8192	Dec. Loss
6-12-39	45	65	85	75	60	100	100	100	73.3	50	55	65	65	40	100	100	100	56.6
9-21-40	35	50	35	35	85	100	100	100	51.6	55	60	75	70	45	75	70	100	63.3

CASE 14. AUDIOMETRIC RECORDINGS BEFORE AND AFTER OPERATION ON LEFT EAR ON JUNE 21, 1939

	Right Ear									Left Ear								
	Audiometer Frequencies								Aver.	Audiometer Frequencies								Aver.
Dates	128	256	512	1024	2048	4096	5792	8192	Dec. Loss	128	256	512	1024	2048	4096	5792	8192	Dec. Loss
6-16-39	50	45	60	55	65	90	100	100	60.	30	40	70	65	70	100	100	100	68.3
9-13-40	35	45	65	60	65	80	100	100	63.3	100	100	85	65	90	100	100	100	80.

CASE 15. AUDIOMETRIC RECORDINGS BEFORE AND AFTER OPERATION ON LEFT EAR JUNE 23, 1939

	Right Ear									Left Ear								
	Audiometer Frequencies								Aver.	Audiometer Frequencies								Aver.
Dates	128	256	512	1024	2048	4096	5792	8192	Dec. Loss	128	256	512	1024	2048	4096	5792	8192	Dec. Loss
6-17-39	60	60	45	45	30	55	40	45	40.	55	55	40	55	40	55	40	35	45.
4-20-40	60	50	40	35	35	55	45	45	41.6	50	45	30	40	30	70	55	50	33.3
10-26-40	65	50	50	35	35	45	35	50	40.	50	40	40	45	30	55	55	45	38.3

CASE 16. AUDIOMETRIC RECORDINGS BEFORE AND AFTER OPERATION ON LEFT EAR ON JUNE 27, 1939

	Right Ear									Left Ear								
	Audiometer Frequencies								Aver.	Audiometer Frequencies								Aver.
Dates	128	256	512	1024	2048	4096	5792	8192	Dec. Loss	128	256	512	1024	2048	4096	5792	8192	Dec. Loss
6-26-39	45	45	50	40	35	70	65	50	41.6	50	45	55	40	55	80	65	50	50.
7-27-39	50	45	45	40	35	65	70	55	40.	40	35	30	20	25	60	75	100	25.
9- 4-40	45	45	60	40	35	55	65	50	45.	55	60	70	45	55	100	100	95	56.6

CASE 17. AUDIOMETRIC RECORDINGS BEFORE AND AFTER OPERATION ON THE LEFT EAR ON SEPT. 8, 1939

	Right Ear									Left Ear								
	Audiometer Frequencies								Aver.	Audiometer Frequencies								Aver.
Dates	128	256	512	1024	2048	4096	5792	8192	Dec. Loss	128	256	512	1024	2048	4096	5792	8192	Dec. Loss
9- 5-39	55	50	45	40	40	75	100	100	41.6	65	60	55	60	45	60	65	100	53.3
9-28-39	50	45	50	45	30	70	100	100	41.6	25	35	20	20	10	75	100	100	16.6
9-13-40	45	50	50	45	35	80	100	100	43.3	55	60	55	40	30	85	100	100	45.

CASE 18. AUDIOMETRIC RECORDINGS BEFORE AND AFTER OPERATION ON LEFT EAR ON SEPT. 15, 1939

	Right Ear									Left Ear								
	Audiometer Frequencies								Aver.	Audiometer Frequencies								Aver.
Dates	128	256	512	1024	2048	4096	5792	8192	Dec. Loss	128	256	512	1024	2048	4096	5792	8192	Dec. Loss
9-14-39	50	45	40	35	35	40	65	50	36.6	100	75	65	60	65	100	100	100	63.3
10-28-39	45	45	40	30	30	50	60	45	33.3	30	35	30	30	20	50	100	100	26.6
12-30-40	45	50	40	25	35	45	60	45	33.3	100	65	50	45	45	65	60	65	46.6

CASE 19. AUDIOMETRIC RECORDINGS BEFORE AND AFTER OPERATION ON RIGHT EAR ON OCT. 20, 1939

	Right Ear									Left Ear								
	Audiometer Frequencies								Aver.	Audiometer Frequencies								Aver.
Dates	128	256	512	1024	2048	4096	5792	8192	Dec. Loss	128	256	512	1024	2048	4096	5792	8192	Dec. Loss
10-19-39	40	60	65	50	55	75	100	100	56.6	25	30	40	20	40	100	100	100	33.3
11-30-40	100	80	90	75	75	100	100	100	80.	30	30	45	25	45	80	100	100	38.7

CASE 20. AUDIOMETRIC RECORDINGS BEFORE AND AFTER OPERATION ON RIGHT EAR ON NOV. 10, 1939

	Right Ear									Left Ear								
	Audiometer Frequencies								Aver.	Audiometer Frequencies								Aver.
Dates	128	256	512	1024	2048	4096	5792	8192	Dec. Loss	128	256	512	1024	2048	4096	5792	8192	Dec. Loss
11- 7-39	30	35	45	45	45	65	65	60	45.	45	45	40	45	35	75	60	100	40.
12-19-40	50	45	35	35	55	70	100	100	41.6	35	35	30	30	35	85	100	100	31.6

TABLE I

RESULTS OF LABYRINTH FENESTRATION OPERATIONS

Case No.	Technic	Time Since Operation	Average Decibel Change	Fistula Reaction	Operative Indications	Partial Loss of Early Improvement	Total Loss of Early Improvement	Complications
9. M. R.	Malleus head not removed	1 Yr. 8 Mo.	13.3 Loss	++	Present except healed perforation in drum		Yes	Middle ear abscess
10. E. G.	Malleus head not removed	16 Months	1.7 Loss	+	Present		Yes	None
11. E. S.	Complete No errors	18 Months	15. Gain	++	Present except healed perforation in drum	Yes		None
12. A. B.	Complete No errors	16 Months	21.6 Gain	++	Present			None
13. W. L.	Complete No errors	15 Months	21.7 Gain	+	Present			None
14. M. H.	Malleus head not removed	15 Months	11.7 Loss	+++	Present except poor bone conduction			Partial temporary facial paralysis
15. R. J.	Malleus head not removed	15 Months	6.7 Gain	+	Present	Yes		None
16. F. R.	Malleus head not removed	14 Months	6.6 Loss	+	Present except scarred drum		Yes	None

TABLE I (CONTINUED)

RESULTS OF LABYRINTH FENESTRATION OPERATIONS

CASE NO.	TECHNIC	TIME SINCE OPERATION	AVERAGE DECIBEL CHANGE	FISTULA REACTION	OPERATIVE INDICATIONS	PARTIAL LOSS OF EARLY IMPROVEMENT	TOTAL LOSS OF EARLY IMPROVEMENT	COMPLICATIONS
17. J. W.	Complete No errors	16 Months	8.3 Gain	+	Present	Yes	.....	None
18. A. Z.	Complete No errors	15 Months	17. Gain	+	Present except scarred drum	Yes	.....	None
19. J. K.	Complete No errors	13 Months	23.4 Loss	—	Present except poor bone conduction	.....	.....	None
20. N. H.	Complete No errors	13 Months	3.3 Loss	+++	Present except much scarred and perforated drum	.....	.....	None

TABLE II

CASES IN WHICH MORE THAN ONE YEAR HAS ELAPSED SINCE OPERATION SHOWING NO HEARING IMPROVEMENT

Case No.	Time Since Operation	Average Decibel Loss Before Operation	Average Decibel Loss After Operation	Average Decibel Change	Fistula Reaction
2.	13 months	51.6	71.	20 loss	—
3.	2 years	55.6	78.3	22.7 loss	—
4.	2 years	41.	43.3	2.3 loss	—
5.	2 years	32.3	45.	12.7 loss	+
7.	2 years	68.3	93.3	25. loss	—
9.	1 yr. 8 mo.	50.	63.3	13.3 loss	++
10.	16 months	36.6	38.3	1.7 loss	+
14.	15 months	68.3	80.	11.7 loss	+++
16.	14 months	50.	56.6	6.6 loss	+
19.	13 months	56.6	80.	23.4 loss	—
20.	13 months	45.	48.3	3.3 loss	+++

TABLE III

CASES IN WHICH MORE THAN ONE YEAR HAS ELAPSED SINCE OPERATION SHOWING HEARING IMPROVEMENT

Case No.	Time Since Operation	Average Decibel Loss Before Operation	Average Decibel Loss After Operation	Average Decibel Improvement	Fistula Reaction
1.	2 yrs. 3 mo.	68.3	36.6	31.7	+++
6.	1 yr. 11 mo.	76.6	60.	16.6	++
8.	14 months	77.3	51.6	25.7	++
11.	18 months	41.6	26.6	15	++
12.	16 months	96.6	75.	21.6	++
13.	15 months	73.3	51.6	21.7	+
15.	15 months	45.	38.3	6.7	+
17.	16 months	53.3	45.	8.3	+
18.	15 months	63.3	46.6	16.7	+

XVI

SURGICAL TREATMENT OF DEAFNESS: REPORT OF EXPERIENCES WITH THE ENDAURAL LABYRINTHINE FENESTRATION*

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AND

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In the past few years the otologic world has seen a great advance in the therapy of deafness by the surgical fenestration of the horizontal semicircular canal.

Historically, surgery for the relief of deafness dates back to 1649, when Rolarius first attempted to improve hearing by rupturing the ear drum. In 1760 J. L. Pettit, and Jasser in 1776, performed a mastoidectomy for the relief of deafness. Again in 1801 Ashley Cooper perforated the ear drum. In 1876 Kussel attempted to improve hearing by the removal of the stapes. A trephine of the promontory was first practiced by Passow in 1892, and this procedure was violently condemned by his contemporaries, Politzer, Denker, Maure, Cozzolineo and Seiberman. In 1904 Barany fistulized the posterior semicircular canal. Floderus in the same year made a fistula in the promontory and covered it with a Thiersch graft. In 1914 Jenkins reported two cases in which the horizontal canal was fistulized. In 1917 Holmgren introduced the technique of opening the superior semicircular canals, attempting to keep the fistula open by allowing the dura and arachnoid to cover it. In 1918 Barany attempted to improve hearing surgically by a two-stage operation in which the fistula was covered with a fat graft. In 1920 Holmgren again opened the promontory and covered it with mucoperiosteum. In 1924 Sourdille, the French surgeon, first performed the many-staged labyrinthopexy.

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The more modern efforts and teachings of Holmgren,[1] Sourdille[2] and Lempert[3] are well known to every otologist.

Much has been said and written concerning these recent developments, especially since the introduction by Lempert, in 1938, of a new one-stage operation performed endaurally. Recently, reports by Campbell,[4] Canfield,[5] Kopetzky,[6] Passe,[7] Shambaugh,[8] Goodyear,[9] and Gugenheim[10] have indicated that although an improvement in hearing by air has been obtained in a number of cases, there is still much to be learned and many problems remain unsolved.

Our experience with the one-stage Lempert endaural tympanolabyrinthopexy dates back to 1938, at which time we performed our first labyrinthine fenestration. Since then we have employed this technique in operating upon 38 cases.

We intentionally did not rush our observations into literature before this because of the uncertainty of the permanency in the improvement in hearing acuity. We have noted recently that cases have been reported in the literature after such short periods as four to six weeks postoperatively, when the critical period of the operation takes place about the sixth or eighth week, at which time there may be a slowing of the fistula response with a consequent diminution in hearing. We have felt that a longer period of observation of the operation and its end result would be more desirable, and in the long run more valuable.

It should be stated at the outset that in a procedure as highly technical and specialized as the fenestration operation for deafness, experience is of great importance. There is no doubt that as the surgeon's technique improves and the surgical time is reduced and the operation expedited, the amount of trauma and postoperative edema will be lessened, and thus better results may be obtained. Lempert[11] recently reported his experiences with 120 cases, 69 per cent of which were restored to practical physiologic hearing. It therefore naturally follows that perhaps with a greater number of cases the percentage of improved cases will be greater.

Of our 38 cases, four were restored to practical physiologic hearing by air conduction, and these have been observed for periods varying from six months to two and a half years. Of the other 34 cases, all the fenestra made into the horizontal semicircular canal closed after periods varying from several weeks to eight months. It is only by continued observation of patients over a period of at least three months that a case may be called successful. As Dr. Crowe[12]

has stated, "No one has the slightest idea why sound waves entering the labyrinth by the abnormal route (a fistula in the horizontal semicircular canal) are so clearly heard and understood. It would be an ideal method for improving the hearing and avoiding the use of mechanical hearing aids in patients whose inner ears and nerve are normal, provided the fistula remains open. New bone forms, the fistula closes and the hearing impairment gradually returns to the preoperative level—at least this has been the experience of others doing this operation. Many operations of this kind have been done on monkeys and histological studies have invariably shown the fistula closed after a few months." Again Crowe states in commenting on the report by E. R. G. Passe, "Here again is a report showing that in true otosclerosis, without cochlear changes, hearing is improved tremendously as soon as an opening is made in the bony wall of the labyrinth. Operations are usually done under local anesthesia, and improvement in hearing is striking but temporary. No method has been found to prevent formation of callus and gradual closure of an artificial opening made in the semicircular canal or the promontory. In some cases new bone grows into and entirely fills the semicircular canal as shown by histologic study, but with proper precaution there is little danger of infection and secondary labyrinthitis. Since operation does not endanger the life of the patient, it is justifiable to continue trying new methods to prevent closure of the fistula."

In addition, we have noted that although our technique has improved our results did not necessarily improve. In some of our later cases, the fistula were opened as large as possible and yet bony closure took place. It may be, however, that a more extensive experience is necessitated. However, the fact that four cases have been completely successful indicates that this is one method by which certain deaf patients may be restored to practical, serviceable hearing by air. Perhaps our method of selecting patients, perhaps the surgical technique, perhaps the method of fenestrating the labyrinth are in need of improvement; yet with continued effort and the combined application of many otologists the ideal result may be obtained.

Our series consists of 38 cases. The technique was the one-stage Lempert endaural fenestration of the external (horizontal) semicircular canal. These cases were carefully selected. All patients were tested numerous times with audiometer and tuning forks. Tests were made for both air and bone conduction with masking as indicated. In addition, vestibular tests were done on each patient and only those with a normal functioning labyrinth were selected.

While the operation is primarily intended for otosclerosis, the diagnosis of otosclerosis is at times a difficult one to make. The number of deafened patients regarded as otosclerotics varies with different authorities. Lempert believes that many more cases of deafness are otosclerotic than one suspects. Actually upon histologic examination only a few deafened patients show true otosclerotic bone changes.

Thus in order to reach a diagnosis and to aid us in the proper selection of cases we adhered to the following routine history, medical and otologic studies. Most cases were hospitalized one week prior to operation so that various X-ray and blood studies could be made.

OUTLINE OF HISTORY FOR THE STUDY OF DEAF PATIENTS

1. Age at which deafness was first noted:
   (a) Onset—
   Sudden
   Gradual
   (b) Did deafness follow—
   Cold
   Influenza
   Pneumonia
   Injury
   (c) Was it related to any other infection or contagious disease?

2. Progression:
   (a) Was deafness gradually progressive?
   (b) Did it became stationary?
   (c) Was the hearing loss all at once and then no change?
   (d) Rate of progression of hearing loss.

3. Tinnitus:
   (a) Onset of tinnitus, before, simultaneous with or after onset of deafness.
   (b) Is tinnitus constant, intermittent, loud or soft?
   (c) Describe noise.
   (d) Location of tinnitus, aurium or capitis.
   (e) Does tinnitus keep patient awake?
   (f) Does noise ever cease?
   (g) Did patient receive any treatment or medicine?

4. Mode of onset of deafness:

   (a) What factor brought deafness to your attention?
   (b) When did you first notice loss in conversational hearing?
   (c) Can patient hear on the telephone?
   (d) Can patient hear watch tick?
   (e) Difficulties in conversation.

5. Childhood:

   (a) Frequency of colds in childhood.
   (b) Age at which tonsils and adenoids were removed.
   (c) Was deafness related to the time of tonsillectomy?
      1. Did it precede it?
      2. Did it follow it?
   (d) History of suppuration from ears—
      1. Acute
      2. Chronic
   (e) History of contagious diseases:
      1. Measles
      2. Mumps
      3. Scarlet fever
      4. Diphtheria
      5. Meningitis
      6. Influenza
      7. Pneumonia
      8. Typhoid
      9. Osteomyelitis
      10. Any other diseases.

6. Family history of deafness:

   (a) Great grandparents { maternal / paternal }
   (b) Grandparents { maternal / paternal }
   (c) Parents { mother / father }
   (d) Other members of the family.

7. Social history:
   (a) History of venereal disease.
   (b) Tuberculosis.
   (c) Smoking.
   (d) Alcohol.

8. Sexual:
   (a) Age of onset of menses.
   (b) Any relation of onset of deafness to menses?
   (c) Does hearing vary at time of menses?
   (d) Menopausal age.
   (e) Pregnancies or miscarriages—their effect on hearing.

9. Occupational:
   (a) Type of work.
   (b) Amount of noise.
   (c) Does patient hear more acutely while at work?
   (d) Is patient able to perform duties?

10. History of trauma:
   (a) Head injury
   (b) Concussion
   (c) Fractured nose
   (d) Fractured skull
   (e) Bleeding from ears
   (f) Injury to ears:
      1. Loud explosions near ear
      2. Gun shot
      3. Telephone percussion.

11. General medical history:
   (a) General health
   (b) Any evidence of thyroid disease
   (c) Any evidence of other glandular trouble
   (d) Cardiac disease
   (e) Hypertension
   (f) Gallbladder
   (g) History of severe illness:
      1. Influenza
      2. Typhoid
      3. Pneumonia
      4. Septicemia
      5. Meningitis

(h) Operations:
1. Nasal
2. Throat
3. Other parts of the body.

12. Ear, nose and throat:
(a) Frequent colds or sore throat
(b) Respiratory infections
(c) Sinusitis—acute or chronic
(d) Ears—acute or chronic suppurative otitis media
(e) Mastoiditis
(f) Pain in ears
(g) Vertigo
(h) Headaches.

13. Teeth:
(a) Infections—cavities—pyorrhea
(b) Unerupted teeth
(c) Edentulous
(d) Disturbed bite
(e) Pain over temporomandibular joint.

14. History of drugs (taken during pregnancy):
(a) Quinine
(b) Salicylates.

15. History of other drugs taken:
(a) Quinine
(b) Salicylates
(c) Arsenic
(d) Poisons—industrial
(e) Alcohol.

16. Allergic history:
(a) Hives
(b) Allergic rhinitis
(c) Serum sensitiveness
(d) Angioneurotic edema.

17. Ménière's Disease:
(a) Vertigo
(b) Dizziness
(c) Nausea and vomiting
(d) Tinnitus
(e) Deafness
(f) Aura.

18. Epilepsy.

19. Neurologic complaints.

20. History of treatment:
    (a) Catheterization and massage
    (b) Politzerization
    (c) Other treatments
    (d) Drugs
    (e) Lip reading
    (f) Did patient ever wear a hearing aid?

GENERAL MEDICAL STUDIES WHILE IN HOSPITAL PRIOR TO OPERATION

1. General studies:
    (a) Heart
    (b) Lungs
    (c) Kidneys
    (d) Blood pressure
    (e) Neurologic studies
    (f) Endocrine
    (g) Urinalysis
    (h) Basal metabolic rate
    (i) X-ray of the temporal bone and skull
        1. Type of temporal bone—pneumatic
                                  diploic
                                  sclerotic
        2. Evidence of pathology in temporal bone
        3. X-ray of the skull for evidence of intra- or extracranial pathology.

2. Blood studies:
    (a) Complete hemograms
    (b) Wassermann
    (c) Blood sugar
    (d) Blood urea nitrogen
    (e) Cholesterol
    (f) Serum calcium
    (g) Serum phosphorus

Note.—Of the 38 cases studied, the medical and laboratory findings were essentially negative with the exception of one patient, a female, who had a high cholesterol of 350, and one who had a minus 13 B.M.R. Both cases were treated medically prior to labyrinthine fenestration.

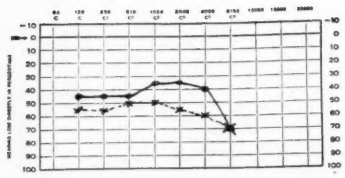

Fig. 1 (Case No. 2). Audiogram made 9-9-40. Three months after the second operation and 21 months after the first operation.

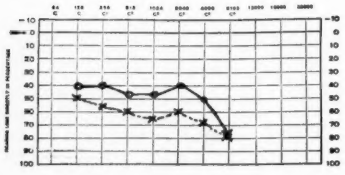

Fig. 2 (Case No. 3). Pre-operation Audiogram 3-1-40.

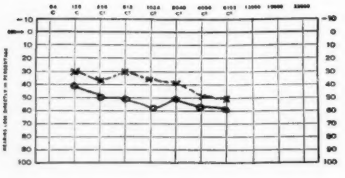

Fig. 3 (Case No. 3). Audiogram made 12-15-40 indicating sustained hearing improvement.

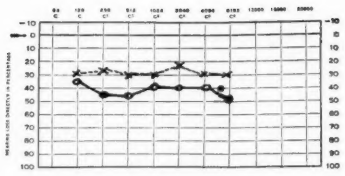

Fig. 4 (Case No. 4). Pre-operative audiogram.

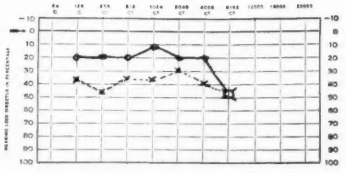

Fig. 5 (Case No. 4). Audiogram made six months later. Note the sustained improvement of hearing. Fistula test is strongly positive.

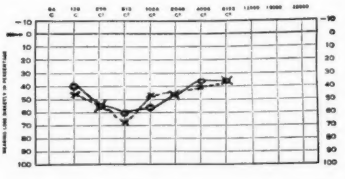

Fig. 6 (Case No. 8). Preoperative audiogram 11-8-38.

3. Otologic studies:

   (a) Hearing tests. (These tests were repeated at least three times at intervals of from several days to several weeks.)
   Functional hearings tests included:
      1. Complete hearing tests by bone and air conduction.
      2. The usual Weber, Schwabach and the Gelle Test for stapes fixation.
      3. Audiograms made both by air and bone conduction. These were done with masking in indicated cases.

   (b) Vestibular tests: These tests included the rotation tests and the mass caloric irrigation.

   (c) Otoscopic observations: These included appearance of the membrana, mobility of the drum membrane and evidence of previous infection.

   (d) Nasopharyngoscopic examination of eustachian orifices and catheter inflation of the tubes for evidence of patency.

To summarize, in the selection of the patient we have tried to use the following criteria:

1. Normal or practically normal appearing drum.
2. Notation of otosclerotic reflex.
3. No evidence of acute or chronic infection.
4. Negative history of suppurative otitis media.
5. Patent eustachian tube.
6. Bilateral progressive deafness.
7. Stapes fixation.
8. Good bone conduction.
9. No evidence of neural involvement.
10. Normally functioning labyrinth.
11. Hearing loss greater than 35 DB by air.
12. The hearing loss by air should not be greater than 70 DB, for the practical improvement obtained is often not greater than 25 — 35 DB, and in order to obtain serviceable hearing the improvement should bring the audiogram into the 35 DB range at least for conversational frequencies of 512-1024-2048.

One cannot be too careful in studying each case individually. Five cases under our observation as possible candidates for surgery revealed, upon thorough examination, that the deafness was due to

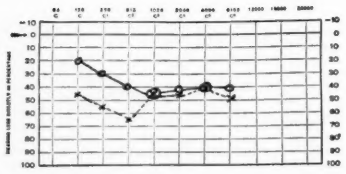

Fig. 7 (Case No. 8). Air conduction audiogram made 15 days postoperatively—at the height of hearing improvement and active fistula.

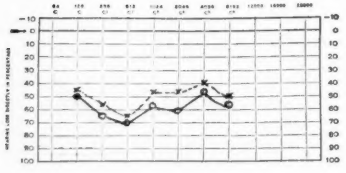

Fig. 8 (Case No. 8). 47 days postoperatively. Fistula response lost. All the gain in hearing lost as soon as this occurred.

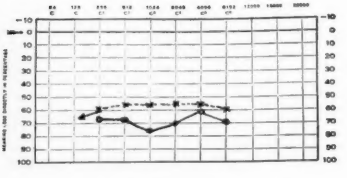

Fig. 9 (Case No. 9). Audiogram (A. C.) made 11-15-38 prior to second operation.

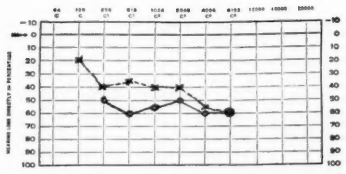

Fig. 10 (Case No. 9). Audiogram made 12-27-38—20 days after the second operation—and at height of the hearing improvement.

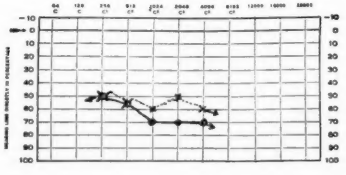

Fig. 11 (Case No. 9). Audiogram made 5 months after second operation. All improvement is lost and there is apparently a progression of the hearing loss.

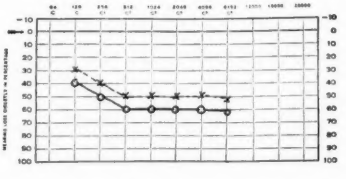

Fig. 12 (Case No. 10). Preoperative audiogram (air conduction).

intracranial neoplasm. These cases were then referred to the Neurosurgical Department.

The 38 patients selected may not have been true otosclerotics, but clinically their tests were such as to lead us to include them in the class suitable for surgery. We have often wondered if it is not possible that in those cases in which the fistula is permanently successful and active that we have accidently struck upon a true otosclerotic capsule. Perhaps there is that possibility that the otic capsule of a true otosclerotic will not regenerate bone as rapidly as that of a patient deafened through some other cause.

The first few cases selected, before we were well acquainted with the limitations of the operation, were those of a quite severe degree of deafness, with hearing loss sometimes as great as 80 — 90 DB and sometimes with quite marked nerve involvement. As a result no permanent or practical improvement was obtained. We learned from experience that in order to expect good results, younger patients and those in whom the hearing impairment was not too extensive should be the ones selected for the operation. In addition, any cases with marked neural involvement are not suitable and therefore are unlikely to respond favorably to labyrinthine fenestration.

Lempert states that there should not be a greater loss than 30 DB for bone conduction in the conversational frequencies.

As a rule, an immediate and striking result in hearing by air is obtained in every patient as soon as the labyrinth is opened and the perilymph fluid escapes. This phase of the operation is very dramatic. Since the operation is done under narcotic local anesthesia, the effect on the patient when he first begins to hear borders on the miraculous. When the flap is placed in position, the hearing for conversational voice is impaired somewhat, although the hearing through bandages and dressings is exceedingly acute as compared to the opposite side. As the postoperative edema of the flap and the surrounding tissues occurs, the hearing again becomes quite impaired so that by the third day it is very poor. After the packing is removed on the fifth to seventh day and the tissue reaction subsides, the hearing again improves and by the end of the second week becomes quite acute. If the result is to be successful this improvement continues. In those cases in which the fenestra closes, the patient's hearing gradually fails as the fistula reaction becomes slower and slower, and by the end of the sixth to eighth week it has completely disappeared.

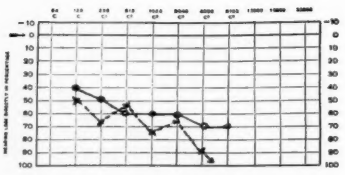

Fig. 13 (Case No. 10). Air conduction audiogram made 7 months postoperatively. Note degree of progression in left ear.

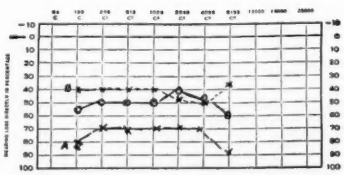

Fig. 14 (Case No. 13). (A) Preoperative curve for left ear. (B) Left ear—19 days postoperatively.

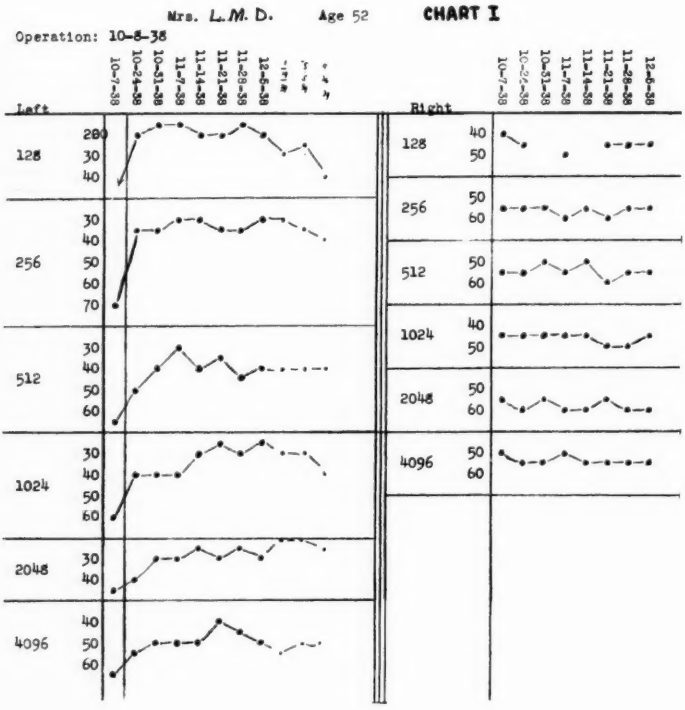

Chart I (Case No. 1). This is a composite chart of the hearing improvement for the various frequencies. Observations extend over a period of one year. Current audiograms reveal that the improvement has been sustained for more than 28 months.

We shall present 17 cases which we feel are illustrative and worthy of reporting. Of the other cases, as the results were unsuccessful, the abstract would be repetitious.

REPORT OF CASES

CASE 1.—Mrs. L. M. D., aged 52, had a history of chronic progressive deafness for the past 10 years and also a bilateral tinnitus. Bone conduction tests were very favorable. The family history disclosed that her father, paternal grandmother, maternal aunt and a cousin all had difficulty with their hearing. Operation was performed October 8, 1938, a left Lempert fenestration. The hearing improvement was noted immediately postoperatively. The usual period of postoperative nausea, vomiting and vertigo occurred. This subsided by the end of the fourth day. The packing was removed on the seventh day. The fistula test has remained positive, and the improvement in hearing has continued for more than two years.

This case illustrates a successful result in an elderly female patient. Chart 1 will demonstrate the sustained improvement in hearing.

CASE 2.—Miss M. S., aged 22, had a history of progressive loss of hearing for the past seven years. There was a bilateral intermittent tinnitus at first, ultimately becoming constant. No history of suppurative otitis media could be obtained. There was no family history of deafness. The general examination revealed no other abnormalities with the exception of the impairment in hearing. The operation was performed December 14, 1938; a right Lempert fenestration. The patient did well postoperatively. The period of nausea, vomiting and vertigo continued for four days. The hearing improvement was immediately noted and was quite marked. The packing was removed on the seventh day, and a prompt and active fistula response was obtained. The hearing has remained good, and to this date, January 20, 1941, the improvement in hearing has been sustained.

In June 26, 1940 (18 months after the first operation) the Lempert fenestration was performed on the opposite or left side in an attempt to relieve the marked and severe tinnitus which was present in this ear. The positive fistula test was also obtained in this ear and is present at the date of this report. The tinnitus was relieved for a few months but has since returned bilaterally, despite the fact that the patient has a bilaterally positive fistula reaction. An audiogram made on September 9, 1940, 21 months after the first operation and three months after the second operation, revealed the following: Despite the bilateral open fenestra, the combined improvement in hearing was no greater for the two operated ears than for one. Furthermore, the hearing loss has definitely progressed despite a bilateral successful operation. In addition, this patient presents the same phenomenon that we have noted in our own as well as in the published cases of others, i. e., a high tone loss which occasionally develops after a successful operation. This is a problem for the physiologist to solve. It is true that the very high frequency losses are of no immediate importance from a hearing standpoint, but from a physiologic standpoint this phenomenon should be explained. Is this the beginning of a neural change? We respectfully call this phenomenon to the attention of the otologists who are doing this type of surgery. This case also points out a very

important fact to us, and while at present it is only a single observation it strengthens a conclusion that we have had in mind, that while the fenestration operation may improve the hearing it does not prevent progression, and the same causative factors may continue to operate and produce further loss of hearing despite the open fenestra. In addition it also illustrates another phenomenon. Here is a case operated upon by the same surgeons, using the same technique, and in which both fenestra remain wide open, and a continued positive fistula reaction occurs bilaterally, whereas in other cases done by the same men with the same technique, or perhaps even better, the fistulas continue to close. Does this not indicate significantly that despite the methods used in opening the fenestra (chisels, burrs, et cetera) and despite the fact that various types of flaps are used, there is still the biologic tendency of bone to heal? We believe that the reason one fistula stays open and another closes lies in the individual type of otic capsule. Perhaps those truly otosclerotic bones which can only be recognized microscopically and not clinically are the ones that remain open. And, further, in a long series of cases carefully selected, the percentage of cases in which the fenestra remain open might be a real indication of the percentage of true otosclerotic cases.

CASE 3.—The history of Mrs. H. S., aged 35, is one of chronic progressive deafness for 17 years. The onset was gradual and the progress insidious. The first symptom was tinnitus. This was intermittent at first but became contant. The patient had many types of treatment without deriving any benefit. There was no definite family history of deafness, but clinically the case seemed to fit into the category of otosclerosis. Operation was performed on March 6, 1940, a left Lempert endaural labyrinthine fenestration. The result to date has been very gratifying. Here we have a constantly positive fistula, relief of tinnitus on the operated side and a definite improvement of hearing.

CASE 4.—The history of Mrs. J. G., aged 27, is one of chronic progressive loss of hearing for 11 years. The patient has been married five years and has two children. The pregnancies apparently did not aggravate the hearing loss. There is marked and rather constant tinnitus. This is present bilaterally. The hearing tests revealed a bilateral conductive type of deafness which was worse on the right side. The bone conduction was very good. The tests for vestibular function indicated a normally reacting labyrinthine system. On June 8, 1940, a right Lempert endaural fenestration of the external semicircular canal was performed. The patient had the usual period of postoperative discomfort. A positive fistula reaction was obtained at the time of the first postoperative dressing. This has persisted to the present time. The hearing improvement has also remained constant. Figures 4 and 5 will illustrate this fact.

CASE 5.—Mr. D. R., aged 19, had a history of progressive deafness and tinnitus for the past six years. The onset of the impaired hearing was in the left ear, which was followed by involvement of the right ear. There was no history of suppuration in either ear. There was no family history of deafness. The hearing tests revealed a chronic progressive type of deafness of the conductive type with fairly good bone conduction. On April 12, 1939, a right Lempert endaural fenestration was performed. In this patient we found a bony ankylosis of the malleoincudal junction. The head of the malleus was separated from the incus with great difficulty. Postoperatively, the patient had the usual stage of nausea, vomiting and vertigo. The hearing test became improved and showed a continued improvement for a period of approximately six months. In this patient we were unable at any

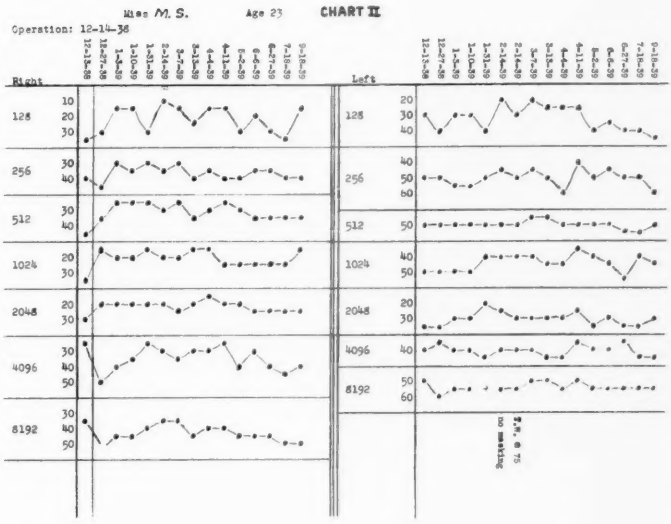

Chart II (Case No. 2). This figure illustrates the sustained improvement in hearing obtained as the result of the successful operation.

time to obtain a positive fistula test on pressure over the fenestra, but he had continued vertigo on any quick motion of the head, either to the right or left, or on turning his body. This persisted for more than a year. At the end of six months the hearing and tinnitus were the same as preoperatively.

In this patient we obtained a marked improvement in hearing for a period of approximately six months. Chart III will illustrate this fact. Despite this hearing improvement, which was not only in the audiometric readings but in the hearing for speech as well, we were unable at any time to obtain a positive fistula reaction. We believe that in this patient we were dealing with a fibrous closure of the fistula or a partial bony closure. Apparently enough of the fistula remained open to produce an improvement in hearing. At the end of six months when closure was complete, the hearing improvement was lost and the tinnitus returned to its preoperative intensity.

CASE 6.—Miss C. F., aged 28, presents a case of chronic progressive deafness of five years' duration, accompanied by tinnitus. There is no family history of deafness and no history of suppurative otitis media. The hearing tests revealed a bilateral obstructive type of deafness with good bone conduction. The vestibular tests revealed a well-reacting labyrinth bilaterally. The operation was performed on June 28, 1938, a right Lempert endaural fenestration. The hearing improved immediately, while the patient was still on the table. There was marked labyrinthine reaction for the first four days; this subsided and the patient made a rapid

recovery. The hearing for conversation was improved, and the fistula test remained positive for about eight weeks, at which time the hearing began to fail rapidly and soon returned to its preoperative level. The tinnitus persisted undiminished from the start, despite the positive fistula test. One year postoperatively, an audiogram revealed the hearing approximately the same as that shown by the first preoperative audiogram. On June 7, 1939, the right side was reoperated and the epithelium lining the cavity was elevated. The labyrinth was again fenestrated. At the site of the old fenestra a small plug of bone was removed, and the fenestra was enlarged. The patient immediately heard again. The flap was replaced. There was a period of nausea, vomiting, nystagmus and vertigo, as at the first operation. The packing was removed on the seventh postoperative day. At this time no positive fistula reaction could be elicited and the patient's hearing rapidly returned to the preoperative level. The improvement obtained by this second operation was very transient.

Case 7.—The case of Miss K. L., aged 38, is one of progressive deafness of 20 years' duration. There is also a history of constant tinnitus in the left ear and intermittent tinnitus in the right. The family history shows that sister, brother and father had impaired hearing. Hearing tests revealed bilateral mixed type of deafness with some impairment in bone conduction. This was probably not a good case to select for this type of surgery. A left Lempert endaural labyrinthine fenestration was performed on June 9, 1938. There was immediate postoperative improvement. There were marked vestibular symptoms during the first few days postoperatively. On June 25, upon her discharge from the hospital, the patient's hearing was very good for conversational speech. The fistula reaction was positive and active. On August 15, approximately eight weeks postoperatively, the hearing receded to its preoperative level and the fistula reaction was lost. In the meantime, the patient had purchased a hearing aid which helped her considerably. On July 20, 1939, one year postoperatively, the patient returned with a new symptom. On lying down, she had marked vertigo and a mixed horizontal and rotary nystagmus. Caloric tests revealed an absence of response to the stimulation of the left horizontal canal. In this patient, possibly an ingrowth of new bone or fibrous tissue had resulted in the loss of function of the left horizontal canal, and upon lying down there was an over-function of the left vertical canal without the compensatory action of the left horizontal. The other semicircular canals on the left side functioned well.

This patient again illustrates a late postoperative complication which probably occurred as the result of disturbing the perilymphatic fluid pathways in the semicircular canals.

Case 8.—Mrs. E. A., aged 35, presents a case of loss of hearing for many years. It was first noted in the right ear, and later the left ear became involved. Tinnitus began at the age of thirteen. There is a definite family history of deafness for the past three generations. The patient was operated upon November 10, 1938. A right Lempert endaural fenestration was performed. There was immediate improvement in hearing of speech, and a marked active fistula reaction persisted for eight weeks, after which there was a gradually diminishing response. There was a loss of the fistula reaction and a return of hearing to the preoperative level. Six months following operation, the patient developed severe attacks of vertigo, et cetera, with symptoms similar to Ménière's disease. The patient was treated and placed on the Furstenberg regime. She still has vertigo on quick change of position.

This case demonstrates several important points: First, the results should not be judged too early. In this patient a spectacular result was obtained early. There was a marked improvement in hearing and a very active fistula test reaction was obtained. This lasted until the eighth week postoperatively. The patient was then apparently well with the exception that her hearing loss returned to the approximate preoperative level. Five months later, the patient began to develop severe attacks of vertigo, tinnitus, nausea and vomiting, which were almost typical of Ménière's disease. Is it not possible that these symptoms resulted from some disturbance of the perilymph fluid pathways as the result of opening the labyrinth?

CASE 9.—The case of Miss I. P., aged 25, is one of progressive deafness for the past 10 years. There is no history of otitis media and no definite family history of deafness. The bone conduction was fairly good. On April 23, 1938, a right Lempert endaural fenestration was performed. There was a very transitory response. The fistula was soon lost, and the hearing tests showed a return to preoperative level. Several months later, on December 7, the opposite (left) side was operated upon (left Lempert fenestration). The immediate hearing response was good. An active fistula reaction was obtained, and this lasted for approximately six weeks. During this time the average improvement for the frequencies 512-1024-2048 was 20 DB. After this time the fistula tests became negative, and the hearing rapidly declined to the preoperative level.

A right labyrinthine fenestration was made in April, 1938. The result following the first operation was very poor. Eight months following the first operation, a labyrinthine fenestration was performed on the opposite or left ear. Here a much better result was obtained. There was a great improvement in hearing and an active fistula response obtained for a period of approximately six weeks. Then the fistula reaction rapidly was lost and with it, the improvement in hearing.

CASE 10.—The case of Mrs. C. S., aged 30, is one of bilateral deafness of 14 years' duration. There is a history of tinnitus and a family history of deafness. Bone conduction was good on the left ear. On February 1, 1939, a left Lempert endaural fenestration was performed. The hearing immediately improved. The fistula response was lost 10 days postoperatively. Hearing quickly returned to preoperative level.

This patient, carefully selected, showed very little if any improvement following surgery. The fistula was carefully made and seemed quite adequate. Yet a fistula response was obtained for only a few days after the packing was removed. There was no evidence of infection or labyrinthitis so that we were unable to account for the rapid loss of the fistula response. This patient was one in whom we noted very little vertigo, nausea or vomiting while we were burring over the fistula. As we explain later, it has been our observation that when there is very slight labyrinthine disturbance upon opening of the labyrinth, the fistula response is soon lost. It will be noted that the audiogram made seven months postoperatively indicated a further progression of the hearing loss. Whether this occurred as a result of the surgery or was a natural retrogression we are unable to say.

CASE 11.—Mrs. N. W., aged 36, presents a case of gradually progressive loss of hearing for the past eight years. There is no history of otitis media. The patient's chief complaint was bilateral tinnitus. The family history revealed that the patient's father and two sisters had impaired hearing. On October 26, 1938,

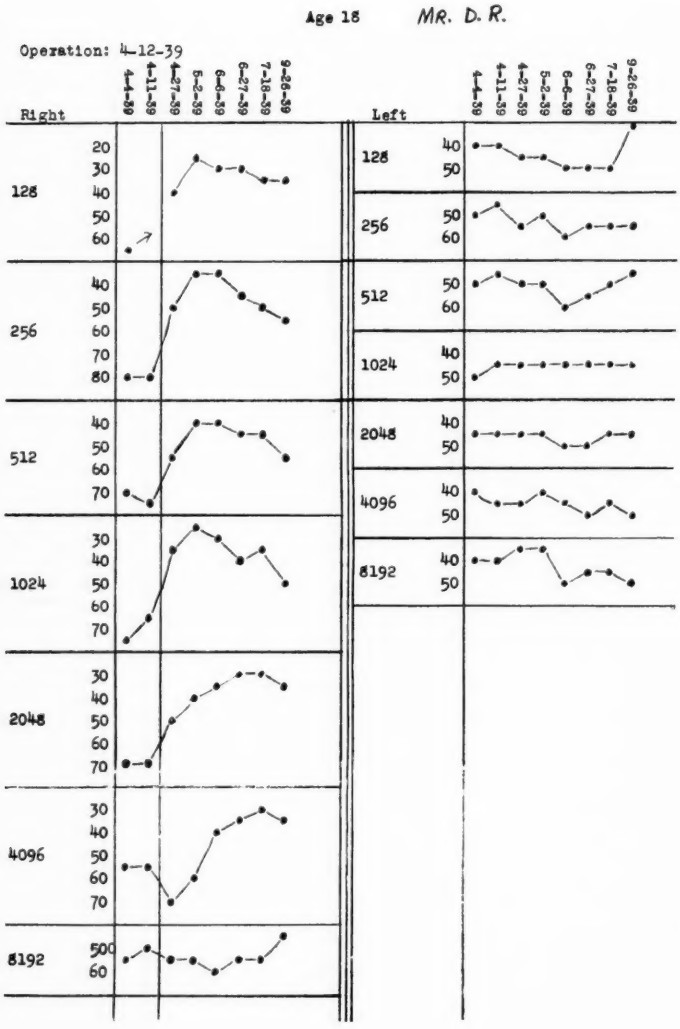

Chart III (Case No. 5). Composite chart illustrating the improvement of hearing in the various frequencies, up to the time that complete elosure of the fenestra occurred.

a right Lempert endaural fenestration was performed. The hearing was improved immediately, postoperatively. There was a gradual reduction of hearing during the convalescent period. When the packing was removed on the seventh postoperative day, no fistula test response could be obtained, nor could it be obtained at any time thereafter. The hearing rapidly fell back to the preoperative level. We may conclude that the result in this case was negative.

Here is another patient in whom very little labyrinthine reaction was obtained when the fistula was first opened. We have learned not to expect favorable results in those patients who do not manifest a labyrinthine reaction.

Case 12.—The case of Miss M. F., aged 28, is one of progressive deafness of five years' duration. There was marked bilateral tinnitus. The hearing tests were good, and bone conduction was good. The vestibular tests were good and active. No history of infection was obtained. On June 16, 1939, a right Lempert endaural fenestration was performed. There was marked postoperative reaction which lasted six days, accompanied by nausea, vomiting and vertigo. The immediate hearing response was good. The fistula response was active. The cavity healed rapidly. By September 1, ten weeks postoperatively, the fistula test was negative, the hearing returned to the preoperative level and the tinnitus recurred.

This patient gave every evidence of a successful result and yet by the end of the sixth postoperative week the tinnitus had returned, the fistula was completely closed and the hearing improvement was completely lost.

Case 13.—Miss E. B., aged 36, had a progressive loss of hearing for the past four years. She also had a bilateral tinnitus. There was no history of otitis media. There was no family history of deafness. Hearing tests revealed a bilateral mixed type of deafness which was worse on the left side. Bone conduction was slightly diminished. The left ear showed a loss of 70 DB at frequencies 512-1024-2048, and it was selected for surgery. On September 1, 1938, a left endaural labyrinthine fenestration was done. There was an immediate improvement in hearing for conversation, and the audiograms showed a definite improvement in the left ear of approximately 30 DB at the conversational frequencies. This continued for seven weeks. An audiogram made three months postoperatively showed that the hearing had receded to its preoperative level. While this patient manifested the typical labyrinthine symptoms postoperatively and showed a definite improvement of hearing, we were unable at any time to obtain a positive fistula test. This case was another that was poorly selected. In the first place, the worse or left ear was chosen, this ear being far below the conversational hearing level. Furthermore there was some suggestion of early cochlear nerve impairment. In cases of this type very little can be expected. Figure 14 will illustrate that even though this patient obtained a temporary improvement of 30 DB in some frequencies, the curve of hearing was still below the 35 DB level of comfortable conversational speech. In other words, at this point it was still necessary to use loud conversation in order to make the person hear. Though no one will deny that this is an improvement, nevertheless the hearing is still not normal. Cases selected for surgery should preferably be those wherein the hearing is not too far below the level of conversational frequencies so that if any improvement is obtained it will bring the hearing curve within the range of conversation.

Case 14.—Mrs. H. J., aged 27, presents a case of chronic progressive deafness and tinnitus for the past 11 years. There was a history of bilateral otitis media

at the age of five years. There was one pregnancy which apparently did not aggravate the hearing loss. The patient's father was hard of hearing. Hearing tests revealed a rather severe type of deafness of the mixed type, with a loss of 65 DB at frequencies 512-1024-2048. The bone conduction was fairly well preserved. On June 14, 1938, a right Lempert endaural fenestration was performed. After the operation no fistula test reaction was obtained. There was a temporary improvement in hearing which lasted but five weeks. This was one of our early cases which was not responsive to this type of surgery.

CASE 15.—Mr. I. B., aged 38, had a history of chronic progressive deafness for 26 years. There was no history of purulent otitis media, but the hearing loss apparently followed an attack of scarlet fever. Numerous treatments of all types were received by the patient without relief or improvement. The hearing loss was at 70 DB for 512-1024-2048. The bone conduction was greatly diminished. This case was poorly selected as it was chosen during the early days of this work when it was thought that only those with a severe type of deafness should be subjected to surgery. On May 21, 1938, a right Lempert endaural fenestration of the horizontal semicircular canal was performed. A positive fistula test was obtained which lasted only two weeks postoperatively. There was a definite improvement for conversational hearing, but by the time the first postoperative audiogram was made, on June 17, the hearing tests showed a very scant numerical improvement. An audiogram made on June 21, one month postoperatively, revealed the hearing to have receded to its preoperative level.

CASE 16.—Mr. R. R., aged 22, presents a history of progressive impairment of hearing for the past six years. There is no family history of deafness nor any history of suppurative otitis media. The hearing loss was of the obstructive type (with fairly good bone conduction). The loss for the conversational frequencies was over 60 DB in the right ear and 50 DB in the left. On February 6, 1940, a right Lempert endaural fenestration was performed. The usual period of postoperative discomfort followed. There was an immediate improvement in hearing for the spoken voice. The fistula test was positive for four weeks postoperatively, after which time we were unable to elicit it. An audiogram made at this time revealed that the hearing had practically returned to its preoperative level.

Here is a patient in whom the hearing improvement was very transient, although surgically the operation was performed with good technique and a good fistula was made. The tympano-meatal flap and other details seemed perfect; nevertheless there was rapid closure of the fenestra. This may have been due either to fibrous or to bony infiltration into the fenestra.

CASE 17.—The case of Mrs. I. T., aged 26, was one of chronic progressive deafness of eight years' duration. There was a bilateral tinnitus. No family history of deafness was obtained. Hearing tests revealed a bilateral conductive type of deafness with a loss of over 40 DB in the right ear and 38 DB in the left for conversational frequencies. The bone conduction was fairly good. On May 6, 1940, a right Lempert endaural fenestration was performed. There was an immediate improvement of hearing for conversational voice. The fistula test was positive for only two weeks. The audiograms made one month postoperatively revealed that the hearing was poorer than prior to the operation. The average loss in the right ear was 62 DB, a loss of almost 20 DB.

POSTOPERATIVE OBSERVATIONS

In order to have a complete picture it might be well to begin with the moment that the labyrinthine fistula is opened and the first drop of perilymph fluid escapes. At this moment the patient usually begins to hear. In addition he begins to complain of nausea and often may vomit. As the burr is moved back and forth over the fenestra there is a definite horizontal nystagmus to the operated side, and this is usually accompanied by vertigo. When the fenestra is completed and after it is covered by the tympano-meatal flap and the paraffin mesh gauze is packed in place, the pressure of the packing may sometimes cause nausea, vomiting, vertigo and a horizontal nystagmus to the operated side. We have noted that in many of our cases that were failures the patients had very little labyrinthine reaction, such as nausea, vomiting, vertigo or nystagmus at the time of the labyrinthine fenestration. This was true despite the fact that the Barany tests were normal preoperatively.

After the patient is returned to bed, the labyrinthine symptoms should persist. The patient usually lies on his good side, in the typical labyrinthine posture. On being addressed he will look toward the quick component of the nystagmus. In some patients the vomiting is quite marked, so much so that in some cases it may be necessary to control this with Nembutal suppositories (rectally) and intravenous administration of glucose and saline. As a rule the labyrinthine vertigo and nausea is controlled with small doses of bromides and phenobarbital, given three or four times daily. These symptoms usually subside by the end of the fourth day. The patient will present a horizontal nystagmus upon looking to the operated side. On looking upward or straight ahead there should be no spontaneous nystagmus.

Beginning on the third day, the patient should be watched carefully for any evidence of serous labyrinthitis. This will be manifested by an increase in the nausea, vomiting, and vertigo. A very important sign is the onset of a spontaneous horizontal nystagmus on looking upward or looking straight ahead. This usually indicates an irritative labyrinthitis. It may indicate (1) that the packing is too tight, (2) the onset of a non-suppurative serous labyrinthitis, and/or (3) the sloughing of the tympano-metal flap which is covering the fenestra.

If any of these signs are noted, the wound should be redressed and all the packing should be removed so as to relieve the pressure on the membranous labyrinth. If a labyrinthitis occurs it may ter-

minate in a dead labyrinth with loss of function of the horizontal canal (localized circumscribed labyrinthitis). Then again, there may be an involvement of all the canals with complete loss of function of the involved side. If this occurs complete deafness will result on the operated side. Fortunately, in our experience there were but two cases of total labyrinthitis and two cases of localized or circumscribed labyrinthitis, which involved only the horizontal canal.

If no untoward complications occur the patient may be allowed to get out of bed on the fifth or sixth day. By the seventh day walking may be attempted. The patient will be quite unsteady at first and may even require assistance for some time. This varies with the individual. Some patients are able to walk perfectly well by the tenth day, while others have ataxia for a period up to three or four weeks.

In those patients in whom the fistula remains patent there is a tendency toward vertigo on quick change of position or rotation of head. This may persist for many months. In some patients with a very active fistula, even yawning, chewing, shouting vigorously, blowing the nose or hard swallowing may produce nystagmus and vertigo.

CONCLUSIONS AND SUMMARY

The despair of the fenestration operation for the improvement of hearing is the closure of the newly created fistula. It is irrelevant whether the scraping method of Holmgren is employed or the Lempert method of using the electric drill with a dull polished dental burr. It is not the mechanical method of producing the fenestral formation that baffles the otologist but the tendency toward osteogenesis.

It has been hypothesized by some that osteogenesis has been retarded or in fact inhibited by the heat which was created by the polishing burr during labyrinthine fenestration. We cannot subscribe to the hypothesis that the heat of a burnishing burr will inhibit osteogenesis. In a recent communication,[13] we reported that the heat created by the smooth, dull burr did not at any time exceed that of three degrees Fahrenheit, as was shown in our experiments. We therefore cannot credit this technique with the success of some of our fistula operations for otosclerosis, nor can we likewise attribute our failures to it. So long as maintaining the patulency of the fistula and the inhibition of osteogenesis remain the crux of the problem there is still much to be learned.

Since 1938 we have performed a total of 38 Lempert endaural operations for the relief of deafness. Of these cases only four were truly successful, in so far as the improvement in hearing is concerned. This can be substantiated by audiometric graphs. Several cases claimed improvement in hearing, but this could not be substantiated by any hearing tests. Incidentally the fistulas in these cases were negative. We must, therefore, always be cautious in evaluating subjective improvement when it cannot be corroborated objectively.

It is highly advisable to watch the patients for a number of months since experience shows that early improvement in hearing may later be lost.

Another point worthy of note is that complications may occur at a much later interval than eight weeks. For example, in the case of Miss K. L., whose left ear was operated upon, it was not until one year after labyrinthine fenestration that this patient developed the extreme symptoms of vertigo and nystagmus upon lying down and turning of the head toward the left. On caloric stimulation it was found that the left horizontal canal had ceased functioning. It may be that in lying down and turning toward the left her left vertical canals are unopposed, thus accounting for her vertigo.

In another patient (D. R.), in whom definite improvement of hearing was noted, we were unable to obtain a fistula reaction at any time. However, this patient up to the present time still becomes quite dizzy on turning his head quickly or upon rising abruptly from a sitting to a standing position.

A third patient (Mrs. A.), upon whom the operation was performed, and in whom the fistula closed with a recession of the hearing to the preoperative level, developed attacks of vertigo, nystagmus, nausea and vomiting almost typical of Ménière's disease, six months postoperatively. These reactions were all delayed, and this must therefore be borne in mind when evaluating the end results.

In several patients the hearing improvement following the surgery was only temporary, and audiograms later showed a further impairment of hearing to a point far below the preoperative level.

It might not be amiss to make mention again of the cases that developed a loss of the high frequencies postoperatively. Is this a forerunner of further degenerative changes either in the cochlear or in the neural mechanism?

SUMMARY OF CASES

Case No.	Name	Age	Sex	Operation	Fistula	Tinnitus	Result
1.	L. M. D.	52	F.	10-8-38	Positive	Relieved	Improved hearing 28 months
2.	M. S.	22	F.	12-14-38	Positive Bilateral	Returned	Bilateral operation. Tinnitus returned despite positive fistula. Hearing improvement sustained.
3.	H. S.	35	F.	3-6-40	Positive	Relieved	Hearing improvement sustained.
4.	J. G.	27	F.	6-8-40	Positive	Relieved	Hearing improvement sustained.
5.	D. R.	19	M.	4-12-39	No Positive Fistula	Returned	Hearing improved for 6 months without positive fistula.
6.	C. S.	28	F.	6-28-38	Fistula closure after 8 weeks	Returned	Re-operated upon. Removal of bony plug. Closure after second operation.
7.	K. L.	38	F.	6-9-38	Fistula closure after 8 weeks	Returned	Labyrinthine disturbance. Loss of function external canal. One year postoperatively.
8.	E. A.	35	F.	11-10-38	Fistula closure after 8 weeks	Returned	Labyrinthine disturbance similar to Ménière's disease 5 months postoperatively.
9.	I. P.	25	F.	4-23-38 12-7-38	Both fistula closed—first operation immediately—second operation in 6 wks.	Returned	Bilateral operation. Both fistulas closed.
10.	C. S.	30	F.	2-1-39	Fistula closed	Returned	Temporary hearing improvement.

SUMMARY OF CASES (Continued)

Case No.	Name	Age	Sex	Operation	Fistula	Tinnitus	Result
11.	N. W.	36	F.	10-26-38	Fistula closed on seventh day	Returned	Very transient hearing improvement.
12.	M. F.	28	F.	6-16-39	Fistula closed on tenth week	Returned	Temporary improvement in hearing and tinnitus.
13.	E. B.	36	F.	9-1-38	Fistula closed on seventh week	Returned	Transient improvement. Poorly selected case. One of the early cases in this series.
14.	H. J.	27	F.	6-14-38	Fistula closed after five weeks	Returned	Early case. Temporary improvement.
15.	I. B.	38	M.	5-21-38	Fistula closed within two weeks	Returned	Too severe a type of deafness—temporary improvement.
16.	R. R.	22	M.	2-6-40	Fistula closed after four weeks	Returned	Transient result.
17.	I. T.	26	F.	5-6-40	Fistula closed after two weeks	Returned	Further impairment of hearing. Rapid closure of fistula.

In several cases we have thought that perhaps the prolongation of the postoperative secretions had some bearing on the fenestral closure.

We tried insufflation of sterile sulfanilamide powder for the purpose of preventing infection and promoting healing. We noticed that the wounds healed much more rapidly. Nevertheless the fistula closed within eight to ten weeks.

Technically, the operation is perfect. By producing the fenestra most patients are able to hear, at least temporarily. We have all experienced the great thrill that comes with the first opening of the labyrinth and noting the immediate response. However, in order that the procedure be practical and serve the greatest number of people, the results should have a greater degree of permanency. This, unfortunately, we cannot guarantee. There should be no danger to the patient from later complications, and he should be allowed ultimately to return to his work. One of our aims is the rehabilitation of the deafened individual. If the patient is going to suffer unending attacks of dizziness, vertigo, et cetera, perhaps it would be better not to disturb his labyrinthine system.

The exact type of patient who will best respond to the operation is by now fairly well established. Yet, in fairness to ourselves as well as to the patient, we are at a loss to make a definite promise as to what the operation will accomplish.

Despite the present state of our knowledge of hearing tests and despite the fact that great care is being taken in the selection of our patients, it is impossible to predict in advance who will be benefited by surgery in so far as the permanent improvement in hearing is concerned. We have considered every type and kind of otosclerosis and have attempted to choose only those patients who come in this category. However, we are all aware that there is great difference of opinion among authorities as to the actual number of true otosclerotic patients.

The few earlier cases operated upon were those of a severe grade of deafness in whom we now realize it is an utter impossibility to expect a favorable result. The cases which later come under our observation were more carefully selected, care being taken that a good degree of bone conduction existed, that there was no evidence of neural involvement, that there was a normally functioning labyrinth, and that the hearing loss by air was not greater than 70 DB. Despite the improvement in the technique employed to restore hear-

ing acuity in these so-called "ideal cases", the results were not as favorable as we had hoped to achieve.

The tendency for the fistula to close is a factor over which at present there is no control. The recent research of Perlman has indicated the prompt tendency for the labyrinth to heal with new bone formation. In our own minds there is a doubt as to whether or not the flap is of any importance in preventing the closure. Parenthetically we may state that we have tried various types of flaps in another series of cases operated upon postauricularly as well as endaurally. The number of fistulas that remain open is small in comparison to those that close. Is it possible that a truly otosclerotic bone has a different morphologic tendency toward osteogenesis? This should lead us again toward making a better diagnosis for otosclerosis.

In opening the labyrinth we necessarily disturb the function of the endolymphatic and perilymphatic systems. Healing of necessity must take place with scar tissue. Is it not possible that in disturbing the fluid pathways we are producing those symptoms of vertigo which we have previously described? In addition, in the healing process the ingrowth of new blood vessels connects the circulation of the middle ear with the inner ear and acts as a pathway for later infections which may ultimately lead to later complications. Here a double circulatory system is converted into one.

Let us also consider the end results. Our goal in opening up a new vista for those afflicted with impairment in hearing is improvement in hearing acuity. Numerical improvement, i. e., 20 DB increase in hearing is unimportant unless it brings the patient's hearing within the 35 DB limit of conversational hearing. Until we accomplish this we have not reached our objective.

A final audiogram that merely shows improvement of hearing is unimportant. Can the patient hear speech? That's the important goal to strive for. If we fail to accomplish this, the surgical procedure with its attendant discomforts, the hospitalization, the long drawn-out course of postoperative treatment, and the loss of time from the daily work-a-day life have been a physical and economic trial and have been in vain.

Finally, we should like to add that it is most encouraging that we are able to report four successful cases. It seems to us that we are on the right track, and we shall continue this work. After all, is there any other form of therapy available at the present time which can boast of such accomplishment? Let us hope that courage, time,

further research of the biology of the otic capsule, and perhaps some surgical modification may lead us to the ultimate solution of this problem.

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XVII

PHYSIOLOGY AND HISTOPATHOLOGY OF THE MIDDLE EAR*

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DEVELOPMENT OF THE MASTOID PROCESS

The usual types of bony mastoid structure are the pneumatic, diploic, and the sclerotic; they have long been the subject of much controversy regarding both their mode of occurrence and their bearing on infectious processes.

The views of Wittmaack on normal and pathological pneumatization have influenced much of the subsequent literature. The development of the pneumatic type of mastoid process seems to be rather generally accepted and substantiated in many laboratories. Eckert-Möbius and Alexander describe findings quite similar to those of Wittmaack.

Pneumatic Type.—One of the primary factors in this development is thought to be the integrity and the growth characteristics of the normal mucosa, which advances from the nasopharynx finally to line the tube, tympanum, antrum and all pneumatic cells of the temporal bone. This process is divided into three stages.

The first stage begins in embryonic and early fetal life with an invasion of the mucosa of the nasopharynx into the middle ear spaces at the expense of the loose embryonic connective tissue which until this time had filled them completely. Thus, at first a cleft is formed which expands until the end of the first year of life, when there is a large cavity extending throughout the tube, tympanum and antrum. This cavity is lined with the invading mucosa and a very thin layer of submucosal connective tissue.

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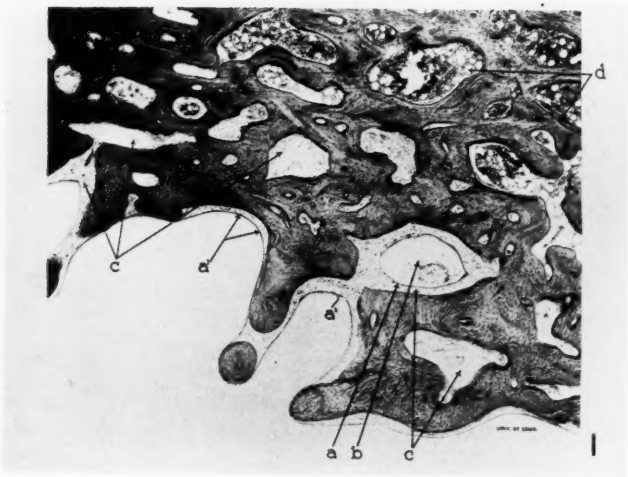


Fig. 1. Photomicrograph: Vertical Section through Edge of Antrum in Infant (High Power). Showing development of pneumatic air cells. *a*, *a'*, lining epithelium of antrum; *c*, connective tissue which has replaced the marrow of the periantral marrow spaces; *d*, unchanged marrow spaces at greater distance from antrum; *b*, pneumatic cell with lining of epithelium and subepithelial connective tissue.

The second stage extends from the end of the first year of life until the age of four to six years, during which time the marrow spaces surrounding the mastoid antrum are transformed into mucosal-lined air cells. This change takes place by a series of steps. First, the thin subepithelial connective tissue invades the periantral marrow spaces, due to erosion of the bony septa, and replaces the marrow. The connective tissue-filled spaces then are lined by extension of the epithelium from the antrum. A simultaneous regression of the connective tissue occurs with the eventual formation of a pneumatic cell lined with mucosa and a thin submucosal layer. With the erosion of the bony septa, many large spaces are formed which later are transformed into smaller compartments as new septa develop. This process continues until the end of the fourth to sixth year when the entire mastoid process is pneumatized (see Fig. 1).

The third stage is simply a further development of the pneumatic cells, keeping pace with the continued growth of the mastoid process in later life.

Nonpneumatic Types.—Wittmaack feels that the nonpneumatic types are also developmental in origin and not generally the result

of infections in later life. This concept is questioned by many authors. According to his findings, when the advancing mucosal lining is undisturbed, normal pneumatization results. If, however, chronic or acute inflammatory reactions have taken place during growth, the subsequent developmental process is modified, and the sclerotic or diploic mastoid process results.

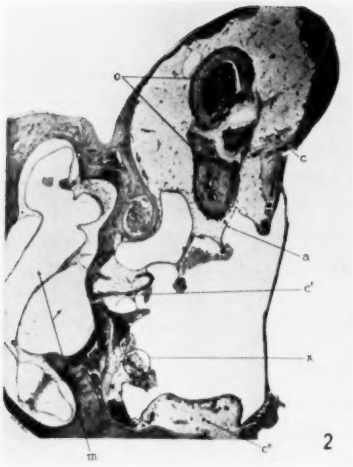
The so-called chronic hyperplastic type is characterized by mild inflammation of the mucosal linings, occurring in fetal life, and is believed to be due to irritation of foreign bodies such as vernix or meconium carried into the middle ear spaces with amniotic fluid. This early, low-grade inflammation tends to slow up the usual rate of regression of the subepithelial connective tissue, so that the ability of the nasopharyngeal mucosa to proceed into these spaces is markedly lessened. Many connective tissue bridges and isolated pockets are seen in the cavities, along with round cell infiltration of the superficial layers of the lining membrane, and many dilated blood vessels (see Figs. 2 to 6).

In postnatal life the same process may continue, due to chronic middle ear infection for which a favorable background has been developed. This is shown in Fig. 7.

With growth the marrow spaces of the mastoid process are slowly replaced by the subepithelial connective tissue as in the normal type, but the mucosa is not able to reach this area due to the marked depth of the subepithelial tissue. Thus the sclerotic mastoid is considered the end result of this process (see Fig. 8).

In milder grades of inflammation, combinations of the pneumatic and sclerotic types frequently are seen. In such cases, the sclerotic bone is always at the periphery.

The second or acute suppurative type is due to acute inflammatory reactions occurring during infancy or early childhood. These infections in healing produce scarring in the region of the mucosal lining (see Fig. 9). Wittmaack believes that in extreme cases this scarring causes a cessation, or at least a marked slowing, of all further attempts at pneumatization; so that if the inflammation occurred in an infant less than a year of age, the marrow spaces of the mastoid process would tend to remain unchanged, and in the adult stage would be the diploic type. There is usually some sclerosis associated with the diploic mastoid process. If the acute infection occurred in a child of approximately two years of age, a half pneumatized mastoid process would be the end-result.



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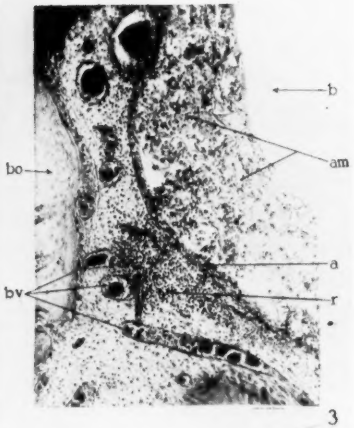

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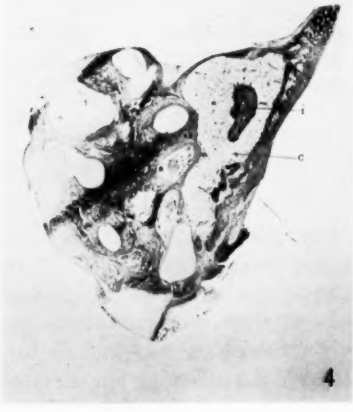

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Fig. 2. Photomicrograph: Vertical Section through Tympanum of Nine Lunar Months' Fetus Showing Retarded Advancement of Epithelial Mucosa. *a*, epithelium which has not reached the attic region; *c*, unregressed embryonic connective tissue filling the attic; *c'*, embryonic connective tissue in region of stapes; *c''*, embryonic connective tissue in region of hypotympanum; *m*, labyrinth; *x*, epithelial cells and debris of amniotic fluid adjacent to lining mucosa. See Fig. 3.

Fig. 3. Photomicrograph: Lining Mucosa of Tympanum (High Power). This is *x* of Fig. 2. *a*, epithelial lining of tympanum; *b*, lumen; *am*, epithelial cells and debris of amniotic fluid; *r*, round cell infiltration of submucosa; *bv*, dilated blood vessels; *bo*, bone.

Fig. 4. Photomicrograph: Vertical Section through Posterior Tympanum of Nine Lunar Months' Fetus. Epithelium has not reached this portion of middle ear cleft. *c*, embryonic connective tissue filling entire posterior tympanum; *i*, short process of incus.

Fig. 5. Photomicrograph: Vertical Section through Antrum in Nine Months' Fetus. No evidence of epithelium in this region. *c*, embryonic tissue completely filling antrum.

That inflammatory reactions can and do occur in fetal and early post-natal life is borne out by various workers. It is reasonable to believe that an extremely altered mucosal lining may affect later pneumatization, as well as the clinical course of subsequent infections. However, Albrecht and Schwartz hold that inborn variations, or constitutional variations in the early mucosal linings, rather than inflammatory reactions of the mucosa, account for much of the variation in adult mastoid structure and later chronic inflammatory conditions.

Numerous observers have felt that following acute infections previously pneumatic mastoids frequently have become transformed into the other types which, in these instances, have no relation to early developmental processes, and that the thickened polypoid lining may be the result of acute infection much the same as in chronic sinusitis.

Cheatle, Mouret and Portmann, on the other hand, feel that the diploic and sclerotic mastoids are normal anatomical variations which may influence inflammatory processes, but which are not necessarily the result of them. A study of large groups of skulls reveals a small number consisting of very dense bone with relatively little diploe even in the surrounding occipital and parietal bones. It is conceivable that in these skulls the usual pneumatic development may be retarded or completely inhibited. However, it is evident that this would occur very rarely.

Practically all writers are agreed on the chronicity of ear infections associated with the nonpneumatic and, particularly, the sclerotic types of mastoid processes.

PHYSIOLOGY AND HISTOPATHOLOGY OF MIDDLE EAR TRACT

The lined cavities of the middle ear tract, i.e., the tube, tympanum, antrum and the pneumatic cells of the mastoid process, are developmentally off-shoots of the foregut and in this respect are analogous to the tracheobronchial tree. Also, their epithelial linings are histologically quite similar and have anatomically adjacent pathways of entrance in the region of some part of Waldeyer's ring. This, according to Wittmaack, could account for the inflammatory involvement of all these tracts in the course of overwhelming infections such as scarlet fever in early childhood.

Eustachian Tube and Tympanum.—The eustachian tube, being the connecting structure between the nasopharynx and the remainder of the middle ear tract, plays a very important role both in the

physiological and pathological processes. Tweedie has summarized the function of the tube as follows:

1. It acts as a normal exit for normal secretions of tympanum.

2. It provides a means of maintaining an equality of air pressure on each side of tympanic membrane for unhindered transmission of sound vibrations.

3. It shares in general congestion associated with acute and chronic nasopharyngitis and thus,

4. Acts as a guard against the introduction of septic organisms.

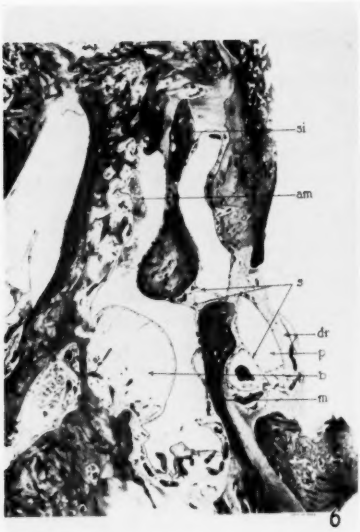
5. As the guard becomes incompetent it constitutes a comparatively easy route for infection of middle ear, and thus,

6. Is a large factor in the occurrence of the major portion of aural pathology.

The auditory tube is shaped like a double trumpet according to Duel. The lumen is smallest at the isthmus which is located at the junction of the bony and cartilaginous portions so that a moderate amount of swelling may occlude the pathway at this point. In infancy the tube is much shorter, relatively wider, and more horizontal in position. The pharyngeal orifice is situated below the level of the hard palate, but in the adult reaches to 10 millimeters above it (MacEwen). It is sometimes yawning in its cartilaginous part, but with growth it normally assumes a slit-like appearance while at rest, due to the lack of supporting cartilage in the lateral wall (Kohlmer). According to the exhaustive researches of Rich, the only time that the tube is patent is during the act of swallowing, when the fibrous lateral wall is pulled away from its opposing medial wall by action of the tensor palati muscle (see Fig. 11). These early anatomical characteristics might well have some bearing on ear pathology, when one considers the usual recumbent position of an infant, especially during frequent feedings.

The noncartilaginous portion of the tube is normally patent throughout in the adult, as it is completely surrounded by bony walls. This bony structure may share in the general pneumatization of the temporal bone with the air cells opening into the lumen of the tube, "cellulae pneumaticae tubae" (see Fig. 12). Anomalies in the size and position of the tube are rare (Keeler). Wittmaack describes a few instances of persistence of the infantile structure.

The submucosal layers consist of loose connective tissue which contains numerous blood vessels, mucous glands and lymphoid tissue.



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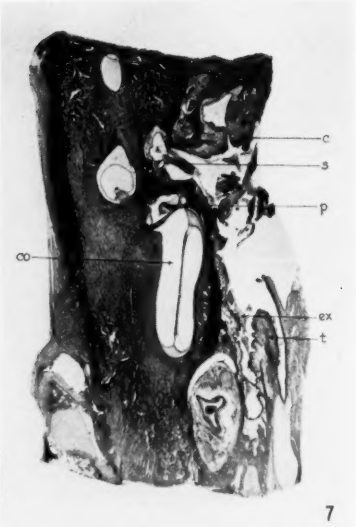

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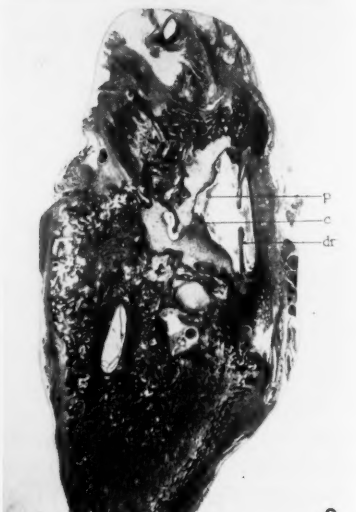

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Fig. 6. Photomicrograph: Horizontal Section through Level of Short Process of Incus of Seven to Eight Months' Fetus. Showing presence of strands and unregressed areas of submucosa, which with increased swelling could effectively separate the attic from the tympanum. *si*, short process of incus; *am*, material in lumen resembling contents of amniotic fluid; *s*, strands of connective tissue lined by epithelium; *dr*, thickened drum membrane containing dilated blood vessels; *p*, Prussak's space; *b*, unregressed embryonic connective tissue mass on medial wall; *m*, anterior process of malleus.

Fig. 7. Photomicrograph: Horizontal Section through Tympanum at Level of Eustachian Tube Orifice of Three Months' Infant. *c*, swollen mucosa showing chronic inflammation; *s*, continued formation of strands and pockets; *p*, swollen mucosa on secondary drum membrane; *ex*, exudate in lumen; *t*, swollen mucosa at tympanic end of eustachian tube; *co*, cochlea.

Fig. 8. Photomicrograph: Vertical Section through Mastoid Process of Adult with Chronic Otorrhea. *c*, connective tissue-filled spaces with no early marrow tissue present; *eb*, sclerotic mastoid tip.

Fig. 9. Photomicrograph: Horizontal Section through Lower Tympanum of One-Month-Old Infant with Acute Suppurative Otitis Media. *p*, purulent exudate; *c*, mucosal lining of tympanum showing swelling and cellular infiltration; *dr*, drum membrane.

Fig. 10. Photomicrograph: Tangential Section through Tube and Surrounding Nasopharyngeal Mucosa.

Toward the pharyngeal orifice the glands are more numerous especially around the deeper crypts formed by the longitudinal folds of the mucosa. Here, also, the lymphoid tissue becomes more in evidence to merge finally into the tubal tonsil (see Fig. 10). The lymph channels, which are somewhat tubular in shape, tend to assume widened sinusoid spaces in the region of the isthmus (Wolf).

The mucosa is made up of columnar ciliated epithelium with many goblet cells. This lining becomes lower near the tympanic orifice and, except for a small area on the floor, becomes normally quite flat and nonciliated in the tympanum, antrum, and in the linings of the pneumatic cells.

The cilia apparently play an important role in keeping the lumen of the tube free of excess mucus and debris by their beating action which is always in one direction, i.e., toward the pharyngeal orifice. This activity, also, would tend to prevent small foreign bodies or infectious material from entering the tube.

Pitman has shown how the changing of the vertical slit of the pharyngeal orifice of the tube into a wider opening during the act of swallowing breaks up the surface tension of any film of secretion that may be stretched across the orifice. This causes the film to collapse and the orifice of the tube is kept open for ventilation of the middle ear.

Interestingly enough there are practically no cilia on the toris tubarius or that part of the tubal lip which extends into the nasopharynx (Lucas).

Working with dyes, Yates found that secretions from the sinuses pass on either side of the convexity of the eustachian cushion to unite again some distance below. However, in cases of sinusitis and chronic ear disease the routes were diverted directly over the tubal orifice providing a constant source of infected material to this region with consequent swelling and blockage of the tube.

He feels that in mild inflammatory conditions, there is an increase in secretion of mucus as well as an accelerated action of the cilia, while in acute infections such as influenza, the cilia on the cells lining the paranasal sinuses and probably those lining the tube, seem to be completely paralyzed. This occurs within a few hours from the onset of "dry rhinitis" due to paralysis of mucous glands, and lasts from three to ten days. It appears that the longer the paralysis lasts, the more susceptible the patient becomes to middle ear infection.

Further protective agents in this region are the possible bactericidal power of mucous exudate and of the local proliferation of white corpuscles and serum. Recently Fenton has shown that the submucosal layer contains histiocytes "which are connective tissue cells, ordinarily quiescent, often near capillaries, provided with vacuoles able to ingest foreign matter." Thus, the extensive connective tissue layer present in infancy may have a considerable protective function.

Since one of the chief functions of the tube is to allow equalization of air pressure on the inside and outside of the drum membrane, any occlusion of this airway produces definite changes in the middle ear tract.

Acute Catarrhal Otitis.—The eustachian tube may be blocked by purely noninflammatory mechanical means, such as pressure of tumors in the nasopharynx, i.e., neoplasms or large adenoids in children. Foreign bodies and scars in the lumen of the tube could also effectively obstruct the airway. Air is known to be absorbed constantly from the middle ear spaces under normal conditions, but is frequently replaced through the tube during the act of swallowing. With obstruction, a continuous negative pressure exists which influences the surrounding tissues. The superficial layers of the lining mucosa respond with a marked dilatation of the blood and lymph spaces, including those of the drum which may show some retraction. Along with the edema of the lining membranes (edema ex vacuo) a sterile serous transudate is produced in the lumen sufficient to compensate for the negative pressure. With the opening of the airway there is a rapid return to the previous normal state.

By far the greatest cause of tubal obstruction is extension of inflammatory processes from the neighboring nasopharyngeal mucosa. Having involved the tubal tonsil, the swelling advances into the tube to involve the superficial layers of the tubal lining. At the isthmus, the increased swelling of the mucosal folds, due to the dilatation of the large lymph spaces known to be in this region, may practically occlude the lumen.

The resultant edema ex vacuo has been previously described. In this case, however, the process is not necessarily self-limited and the infectious irritant may cause greater swelling, exudation and bulging of the drum. Frequently the process goes on to acute suppurative otitis media.

Chronic Catarrhal Otitis.—As the result of continued swelling and hyperplasia, or scarring of the lining, and the subsequent inadequate passageway through lumen of the tube over long periods of

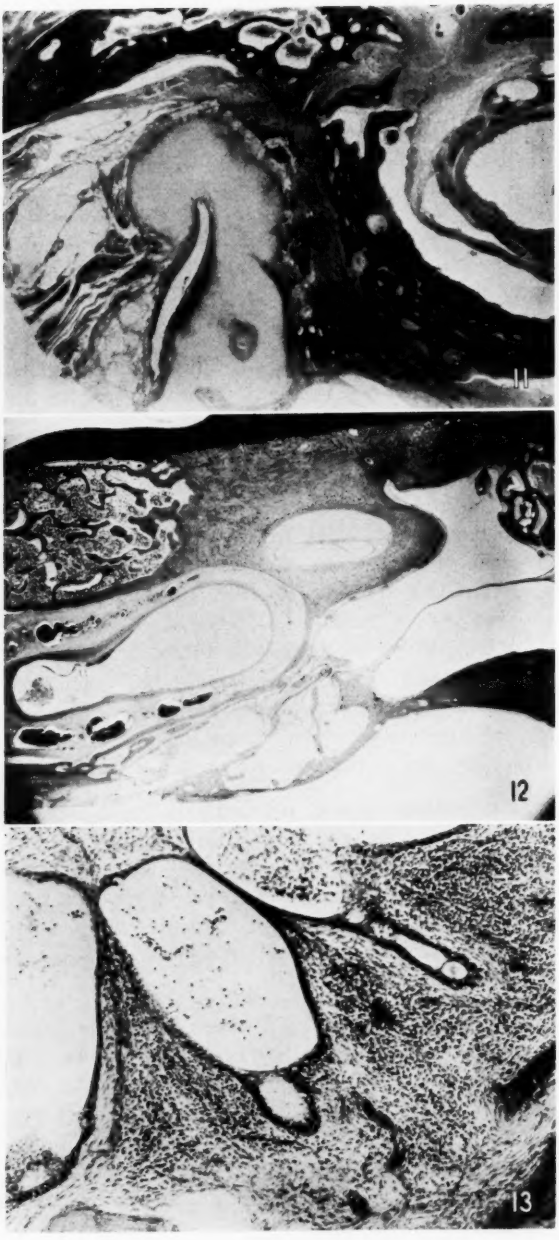
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Fig. 11. Photomicrograph: Vertical Section of Eustachian Tube Near Pharyngeal Orifice.

Fig. 12. Photomicrograph: Horizontal Section through Tympanum at Level of Tube.

Fig. 13. Photomicrograph Showing Newly Formed Strands and Adhesions in Lumen (High Power).

time, the acute catarrhal condition may become chronic. With partial absorption of the fluid by the lining membranes of the cavities there remains a thick mucoid exudate, especially around the region of the ligaments and small depressions, i.e., the round and oval windows. The inflammatory reactions in the superficial layers of mucosa may result in adhesions of neighboring areas, and frequent strands and sheets of fibrous connective tissue advance along the mucoid residue (Dickie; see Fig. 13). These new formations later become covered with the common epithelium of the tympanum. This may be followed by shrinkage, atrophy, and calcification and even ossification of the involved structures (Politzer). The tympanic cavity may thus be converted into a series of more or less isolated compartments with ankylosis of the ossicles (see Fig. 14).

ACUTE SUPPURATIVE OTITIS MEDIA INCLUDING MASTOID INVOLVEMENT

Comparison with Changes in Acute Catarrhal Otitis.—The pathological changes seen in acute suppurative otitis are similar to those of acute catarrhal inflammation except that they are more marked. The degree and extent of involvement depends upon several factors, i.e., (1) local resistance of the lining membranes, (2) general resistance, (3) virulence of the organism, and (4) type of bony mastoid structure.

In the normal development the lining membranes of the tube, tympanum, and mastoid cells are found to be continuous, and in acute infections all of these structures often may be markedly involved soon after the onset. This rapid process, often observed clinically, was well demonstrated by Meyer in his experiments on apes. A short time after one introduction of a strong irritant into the tympanic cavity, he found an acute inflammatory reaction in the lining mucosa of the entire middle ear tract.

The swelling of the tissues in acute suppurative otitis, although similar to that found in acute catarrhal otitis, may become much greater, due to the fact that the marked irritation of an infectious agent has been added to the purely mechanical edema ex vacuo of the latter condition. In extreme cases, the swelling may be so marked as to occlude the lumina of the mastoid air cells, and may reach as high as 80 times the original thickness of the lining, according to Scheibe.

The early edema may be a favorable factor in the prevention of advancement of purulent exudate into the more distant compart-

ments of the middle ear tract. This would be particularly true in infants with incomplete regression of the submucous connective tissue, and where there is already present a thickened lining, caused by previous chronic inflammations. Watkins-Thomas and Yates have clearly outlined the process by which the swollen mucosa successively acts to block the further progress of infectious material along this tract.

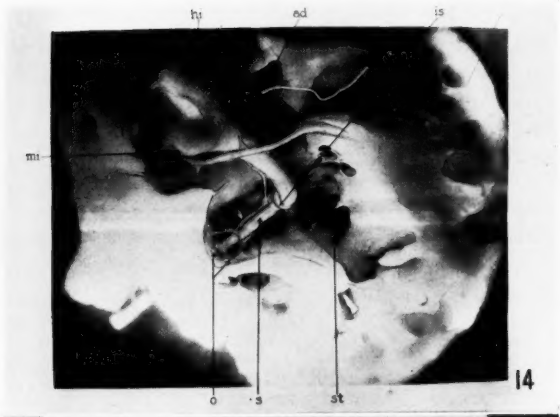
In the following explanation it is obvious that the process is primarily one of occlusion of lumina of the middle ear cavities, and does not take into consideration extension of infections via the mucosal linings.

It has already been shown how extensions of inflammation from the nasopharynx may more or less completely occlude the tube at the region of the isthmus, and thus purulent exudate in the region of the pharyngeal orifice could be excluded effectively from the middle ear tract. However, if this barrier is not sufficient, pus would next reach the tympanum, but the advancement might again be arrested either by drainage through the drum membrane, or by the valve-like action of the swollen mucosa separating the tympanum from the attic, i.e., the swollen mucosa forming the folds of Tröltsch as well as that lining the other structures found at this level of the tympanum. The obstruction is often made more effective by the presence of accessory folds and strands of tissue, which are involved in the general inflammatory reaction.

Should the infectious material break through this constricted area it reaches the upper compartment or attic. Here its further progression may be halted by swelling of the membranes lining the narrow aditus as well as those covering the flared-out posterior ligament. After having passed through the aditus into the antrum, the swollen membrane lining the antrum would tend to occlude the ostia of the periantral cells and thus to prohibit passage to more remote mastoid air cells.

Having once reached the tip cells, however, these valves now just as effectively would prevent drainage from the tract. The result of inclusion of pus by these processes will be discussed in detail in conjunction with the associated pathology of the respective anatomical location.

Infection in a Well-pneumatized Temporal Bone, Osteitis, Coalescent Mastoiditis.—The pathology of acute otitis media and mastoiditis with marked purulent exudate, occurring in a normally lined,



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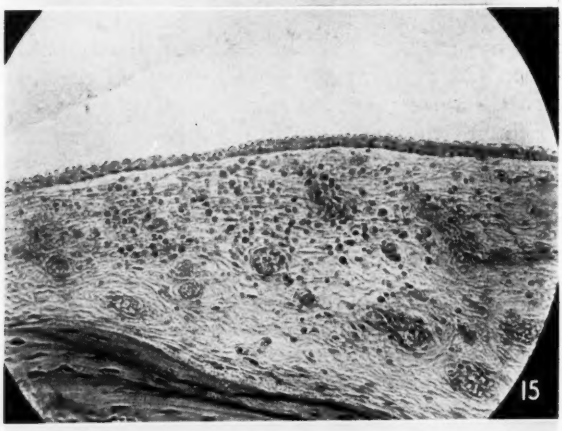

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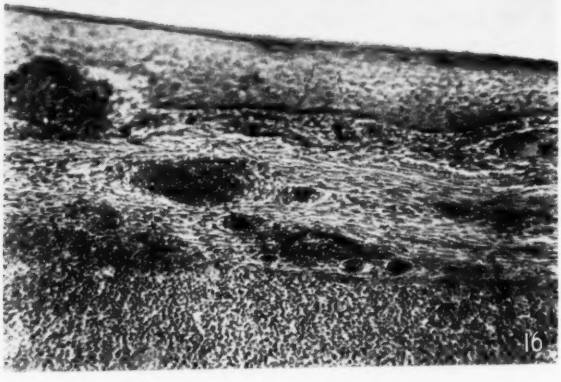

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Fig. 14. Tympanic Cavity Showing Many Fibrous Bands. *bi*, head of incus; *ad*, strands in vicinity of aditus; *is*, incudostapedial articulation; *st*, region of sinus tympanicus; *s*, stapes; *o*, oval window; *mi*, strands in region of incudomalleolar articulation.

Fig. 15. Photomicrograph: Section through Acutely Inflamed Lining Membranes (High Power).

Fig. 16. Photomicrograph: Cross-section of Acutely Inflamed Drum Membrane (High Power).

well-pneumatized temporal bone of a previously healthy individual, is first described.

The initial stage of infection is characterized by increased local blood pressure which is relieved to some extent by swelling of the surrounding tissues, according to Stewart and other workers. The cells of the mucosa, as well as those of the submucosa, become swollen and the former change from a flattened to a more cuboidal shape, although the nuclei remain the same. In the region of the dilated vessels there is a rapid perivascular infiltration with a subsequent migration of cells into the deeper layers as well as toward the surface. Soon the clear fluid exudate in the lumina of the cavities tends to become more purulent (see Fig. 15).

When there is ready exit for the purulent exudate little destruction of the adjacent structures is noted. The similarity of this "simple cell suppuration" to an acute suppurative maxillary sinusitis has been noted by Scheibe. However, in the former case a much more complete and effective blockage of exudate may take place. The effects of increased pressure are often first noted in the drum membrane, which shows a marked bulging and cellular infiltration of all of its layers. There is also frequently erosion accompanied by escape of pus through the perforation (see Fig. 16).

Bulging or sagging of the posterosuperior portion of the external canal wall frequently accompanies acute suppurative infections involving the attic and antrum. This is especially true in infants and young children as the drum membrane, although firmly bound down to the sulcus of the annulus tympanicus, is relatively free in the region of the notch of Rivinus. Here the submucosa of the pars flaccida of the drum becomes continuous with the subcutaneous layer of the posterosuperior canal wall. Marked inflammation of the pars flaccida involving all the layers of the drum tends to extend upward and backward along the common subepithelial layer with the resultant swelling of the posterosuperior canal wall. Inflammatory involvement of this posterior region also may take place directly through the thin cortex over the antrum in infancy (see Fig. 17).

When the infectious products have reached the region of the antrum and mastoid cells, the lining membranes may become so edematous that the lumina are completely filled, or at least the ostia of a cell or a series of cells are completely occluded. Thus, with the production of more exudate under pressure in these closed spaces, the so-called "danger zone" of Scheibe is reached, usually after the first week (Fig. 18). Since this lining membrane is the endosteal

layer, the infection and pressure produce an osteitis. Irregular bony edges are seen with osteoclasts present in Howship's lacunae (see Fig. 19). When this process is present on both sides of bony intercellular septa, there is rapid coalescence of cell cavities which may involve a great portion of the mastoid process with the formation of many bony sequestra (see Fig. 20). On the other hand, it may be limited to a small group of affected air cells. Areas of bony erosion frequently are seen surrounding the blood vessels which enter the bone from the muco-endosteum, with many osteoclastic cells lining these perivascular spaces.

Not all bone destruction proceeds directly from the infected muco-endosteum, but often takes place in the neighboring bone structure itself, through activity in the haversian and in Volkmann's canals. Here there is a collateral reaction to the neighboring stimulus with rapid production of loose connective tissue to fill the bony channels completely. The periphery is often entirely lined with osteoclasts in Howship's lacunae (see Fig. 21). The contents of these canals show no cellular infiltration until the lumen of the air cell is reached, when there is secondary involvement of the connective tissue from contact with the inflammatory cell lining.

Many workers feel that halisteresis, or the absorption of mineral salts from the bone by body fluids, plays an important role in preparing the bone for further destruction by osteoclastic activity. The presence of fibrillation of bone margins without the presence of any cells to account for the change is thought by Stewart and others to be in support of this decalcification process.

Resorption of bone around swollen Sharpey's fibers of the cortical layers of the mastoid process was noted by Scheibe. He believed that the resultant sponge-like change in the compact bone was due to progression of the infectious process toward the surface. On the other hand, Krainz has described similar changes in bone taken from entirely normal temporal bones where no infection was present. It is often difficult to determine whether the lightly stained bone edges are in process of having mineral salts laid down or absorbed. Future research may lead to more uniformity of opinion on this subject.

The location of bony destruction depends to a large extent upon the areas of the middle ear cavities that are effectively blocked.

1. Bony erosion most frequently occurs in groups of pneumatic cells of the mastoid process. With the extension toward the zygoma or other cortical surface, the erosion may reach the periosteum with the production of a subperiosteal abscess, while if it involves a group

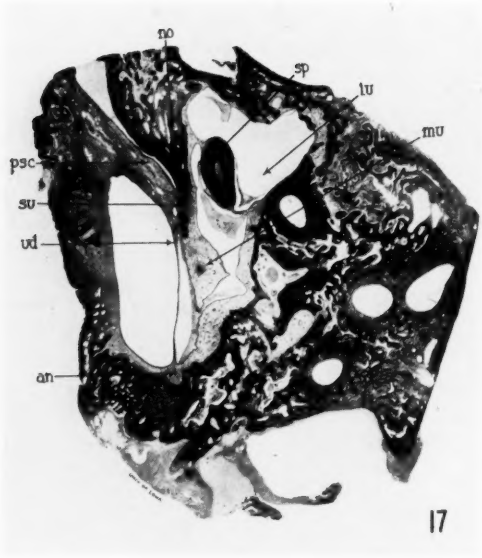
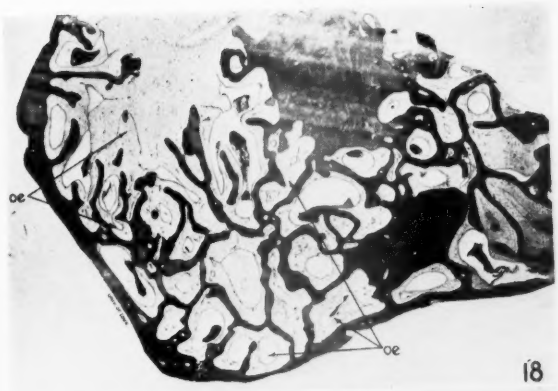


Fig. 17. Photomicrograph: Vertical Section through Posterior Tympanum in Ten Months' Infant. Showing swelling of lining membranes of upper tympanum continuous with swelling of subcutaneous layer of posterior canal wall. *no*, section through notch of Rivinus; *su*, swollen submucosal lining continuous with swelling of posterior superior canal wall; *psc*, swollen posterior superior portion of external canal wall; *ud*, section of swollen pars flaccida of drum; *an*, fibrous layer of drum firmly inserted in sulcus of annulus tympanicus; *mu*, swollen tympanic lining membranes extending medial to drum; *lu*, lumen of posterior tympanum; *sp*, short process of incus.

Fig. 18. Photomicrograph: Vertical Section through Mastoid Process of Six-Year-Old Child with Acute Mastoiditis and Meningitis. *oe*, edematous lining membranes which practically fill the lumina of the air cells.

Fig. 19. Photomicrograph: Bone Erosion in Acute Suppurative Mastoiditis (High Power). *ic*, intercellular bone; *h*, Howship's lacunae; *oc*, osteoclasts near Howship's lacunae.

Fig. 20. Photomicrograph: Sequestra in Acute Suppurative Mastoiditis.



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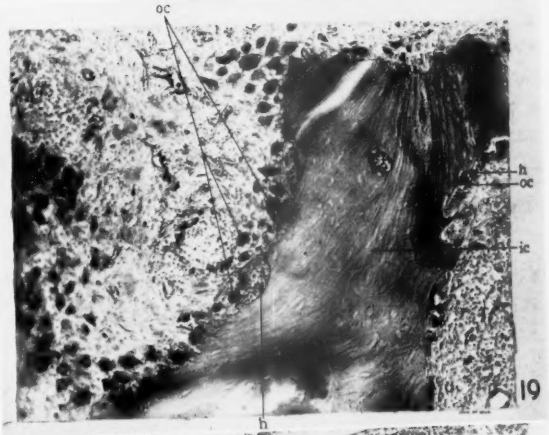

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of air cells near the tegmen antri, the meninges or brain of the mid-fossa may become involved. The fistulae through the cortex are usually smaller than those through the tegmen. Scheibe feels that this is due to the fact that when the infectious process reaches the meninges and brain, the pressure is not as greatly reduced as when it reaches the subcutaneous tissues over the mastoid process. Posteriorly, the lateral sinus and structures of the posterior fossa are liable to invasion, while medially, infection of air cells around the facial canal and, rarely, around the semicircular canals, may lead to involvement of these structures. With complete pneumatization, extension may occur along a chain of pneumatic cells, either above or below the superior semicircular canal, which leads inward along the posterior surface of the temporal bone to the petrous tip (Kopetzky-Almour; see Fig. 22). In this location the chances for rapid exit of the pus are slight, and further involvement of the surrounding deep structures may readily take place. Blockage of pus in the antrum proper and immediate periantral cells is seen chiefly in infants and young children, as the result of breaking through the thin cortex or suture lines, resulting in a subperiosteal abscess.

2. The inclusion of pus in the attic is much less frequent with the resultant extension through the tegmen, along the zygoma, or medially toward the petrous apex, or rarely through the thin wall of the facial canal.

3. Areas of erosion in acute mastoiditis are even more rare in the walls of the tympanum and bony tubal region. With pus under pressure here, rupture of the less resistant drum membrane usually provides adequate drainage and consequently a low incidence of internal ear involvement is seen. However, in infants with unregressed embryonic connective tissue, masses of polypoid tissue may lie medial to the drum (see Fig. 17). Also a much thickened drum may be present, associated with chronic catarrhal conditions. During an acute suppurative infection the drum membrane in these cases may be more resistant to pressure and marked inflammatory involvement. Furthermore the usual myringotomy would afford inadequate drainage because of the more rapid closure of the wound by the polypoid material near the drum.

The peritubal air cells may become occluded with advancement of infection along their chains inward toward the petrous apex. During the process of pneumatization, chains of cells lead backward behind the facial nerve toward the mastoid tip with the possibility of early involvement of the facial nerve accompanying infections of the tympanum in childhood.

Reparative Processes in Mastoiditis.—With the recession of the infectious process, through relief of the excess pressure or other factors, there is a diminution of the rapid exudation and a marked tendency for healing. The integrity of the mucosa is an important factor in the absorption of the exudate, and governs largely the degree of healing that will take place. In mild cases where there has been little destruction of the mucosa, with healing there is marked shrinking with more or less fibrosis of the mucosal lining but usually the lumen resumes its previous contour.

When there are large denuded areas of lining mucosa in the tympanic cavity, the tendency toward chronicity is marked. Very soon after the healing process begins, fibroblastic tissue and small capillaries are seen to advance from the submucosa through breaks in the epithelium, and there is rapid organization of the exudate filling the lumen (see Fig. 23). With further healing there is some fibrosis and shrinkage of the connective tissue filling the lumen, especially of the larger cavities, with only small spaces lined with epithelium remaining in the so-called pearl necklace appearance (see Fig. 24). In this connective tissue matrix which is really a proliferation of the endosteal lining of the air cells, reticular bone is laid down and in small pneumatic cells or groups of cells whose lumina may be completely filled with this early membranous type of bone (see Fig. 24). Even in the early stages of the disease, bone formation is often seen at the periphery of a destructive process (see Fig. 25). As in bone repair in other locations, early marrow tissue finally may replace the loose connective tissue between the trabeculae of the reticular bone so that areas of diploic bone may be the end result of the coalescent type of mastoid involvement.

Along with the reticular bone formation and especially where there is slower healing or continued chronic infection, lamellar bone is laid down. Thus at the edge of the affected bone, layer upon layer of solid new bone is seen with many osteoblasts along the periphery (see Fig. 26). This would result in a thick, eburnated wall of bone, the thickness of which would depend on the amount of destruction that had taken place.

In general, the pneumatic type of mastoid process with normal thin lining lends itself more quickly to rapid bone destruction and fairly rapid healing, depending on the degree of involvement. This is also more or less true where the lining has been scarred by previous acute infection. However, in the thickened hyperplastic lining seen in the sclerotic type, the reactions have a tendency to be less severe

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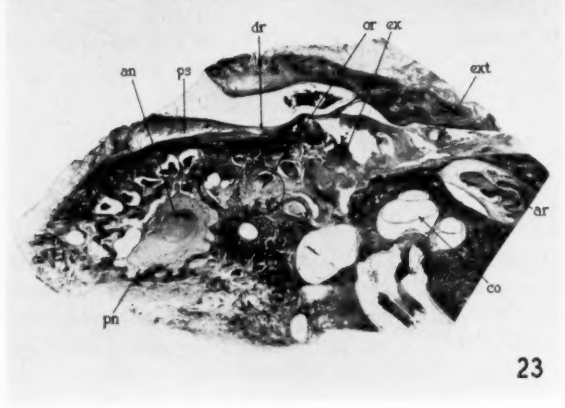
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Fig. 21. Photomicrograph: Bone Surrounding Lumen of Air Cell (Medium High Power).

Fig. 22. Photomicrograph: Horizontal Section at Level of Tendon of Tensor Tympani through Pneumatized Adult Temporal Bone. Showing chains of air cells leading from antrum toward petrous apex.

Fig. 23. Photomicrograph: Horizontal Section through Temporal Bone of Five-Months' Infant with Acute Suppurative Otitis Media. *ext*, purulent exudate filling tube; *ex;* purulent exudate in anterior tympanum; *or*, beginning organization through defect in epithelial lining; *pn*, organization and shrinkage of contents of antrum with formation of "pearl necklace" spaces; *dr*, swollen drum; *ps*, extension of swelling of drum to involve posterior superior region of external canal wall; *co*, cochlea; *an*, lumen of antrum filled with organized exudate.

and the acute infections usually appear unable to penetrate into the deepest layers of the endosteal submucosa with comparatively less rapid destruction (Wittmaack). Nevertheless, there is a tendency for continued chronicity and isolated areas of infection, which may produce serious complications because the infectious material is unable to find an exit through the cortex (see Fig. 27).

Hemorrhagic Mastoiditis.—Another type of acute pathology is seen associated with the extremely virulent organisms such as the hemolytic streptococcus or influenza bacillus. This is known as vascular route infection or the hemorrhagic mastoiditis.

In contrast to the usual acute suppurative type the lining membranes are not so markedly swollen and the cells of the mucoendosteum are quite well outlined, according to Beck. The submucosa, however, contains a large number of greatly dilated vessels with many areas of hemorrhage. The lumen of the air cells are filled with serosanguineous exudate unless secondary infection is present. The bony septa usually show no marked signs of erosion (Kopetzky). These cases frequently have an early blood stream infection with a high percentage of involvement of the lateral sinus due to the extension along the small venous channels.

Acute Osteomyelitis.—Fortunately, osteomyelitis or infection of the bone marrow is a rare complication of acute mastoiditis. In the completely pneumatized mastoid bone, there is a relatively small amount of diploic bone which would be subject to invasion. However, in the mixed and diploic mastoid processes, frequently very little pneumatization has taken place, even in the adult. The periantral region is a favorable anatomical site in infants under one year of age, since the separation of the antrum lumen and the surrounding marrow-containing spaces is not wide. Even here there is usually a high layer of unregressed subepithelial tissue which is believed to be an efficient barrier against deep invasion of infectious products (Wittmaack). In addition to this, infection in the lumen would cause even greater swelling of the lining membranes with possibly more protection in the average case against deep invasion. Meyer feels that this swollen subepithelial layer has a strong protective function in infancy. Proved osteomyelitis of the temporal bone has been observed only once at the University Hospital during the past seven years. This occurred in a ten-month-old infant following scarlet fever.

Where there has been partial pneumatization, i.e., marrow spaces remaining in the mastoid tip, either during the course of develop-

ment or when found in the adult bone, there is usually a more or less thick solid bony separation between the air cells and marrow spaces as well as a layer of subepithelial connective tissue (see Fig. 9).

Other sites of possible marrow space infection are the zygomatic regions, squamosa of temporal bone and incompletely pneumatized petrous apex. Occasionally the infection occurs in the diploic bone around the bony labyrinthine capsule.

The virulent infection, once having invaded the marrow spaces, spreads rapidly, and complications due to invasion of surrounding structures are common, as well as the spread of the osteomyelitis to the adjacent skull bones. There is an accompanying thrombophlebitis of the osseous veins with often a serious general septic infection. It is apparent that in this condition the mortality rate is high. In infection of the nonpneumatized diploic petrous apex, where there has been invasion from the adjacent ear cavities or sphenoid, such a pathological process is probably responsible for the short stormy clinical course terminating in death.

Necrosis.—Rarely in markedly aged or debilitated individuals suffering from tuberculosis, syphilis, etc., as well as in children with scarlet fever, necrotic areas of the temporal bone may occur. The usual marked swelling and inflammatory reaction of the lining membranes are not seen, but rather a destruction of much of the tissues in the affected region occurs. Areas of necrosis of the entire mucosal linings as well as the drum may take place. There is death of bone, probably due to inadequate nutrition without the usual evidence of blockage of pus under pressure. Scheibe feels that, except when necrosis follows osteomyelitis, it is due to poor resistance found in weakened bodies (see Fig. 20).

CHRONIC SUPPURATIVE OTITIS INCLUDING MASTOID INVOLVEMENT

Etiological Factors.—Chronic suppuration may be the result of a number of etiological factors. Chronic inflammations with thickened lining membranes before and soon after birth have been described by Wittmaack and others, and are thought by them to favor continued suppuration. Also chronic infection may result from a low grade infection superimposed on a chronic catarrhal otitis media in the adult, as discussed under that heading. It is a frequent sequel of acute suppurative otitis media and mastoiditis, accompanying acute general infections such as scarlet fever, during which the lining epithelium has been largely destroyed with resultant large areas of thickened and denuded submucosa having poor absorptive power.

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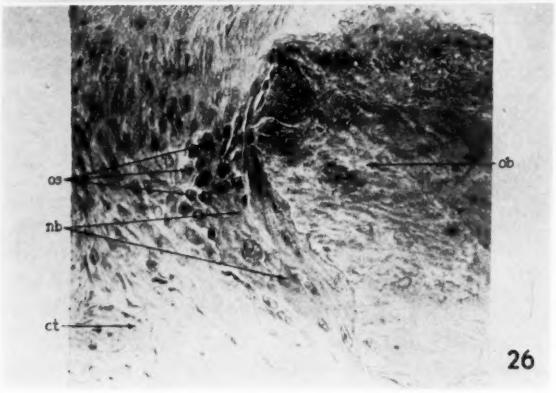
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Fig. 24. Photomicrograph: Section through Lumen Following Acute Suppurative Mastoiditis.

Fig. 25. Photomicrograph: Bone Formation and Bone Destruction (High Power). *bo*, osteoclasts in Howship's lacunae; *nb*, osteoblasts at edge of new bone being laid down in connective tissue matrix (*ct*).

Fig. 26. Photomicrograph (High Power). *ob*, old bone; *nb*, new bone with osteoclasts incorporated in it; *os*, osteoblasts at periphery laying down osseomucin; *ct*, connective tissue matrix.

With the continued stimulus from the chronic suppuration, a marked hyperplasia of all the membranous linings may result (see Figs. 27, 28).

The lumina of the air spaces are irregular with strands and isolated pockets, and deep recesses which may be lined by epithelium and continue to show signs of active inflammation in spite of general subsidence elsewhere. The granulations and hyperplasia of the lining may become so large as to fill up the lumen of the tympanum completely and at times protrude through the drum (see Fig. 29).

The lining mucosa of the tympanum and even antrum may react to the chronic mild irritation by developing definite columnar ciliated type of epithelium. This has been noted by Brunner and others (see Fig. 30). Wittmaack was able to produce this change artificially in laboratory animals, by long-continued, mild stimulation. There is more or less fibrous connective tissue present throughout the lining with a variable but definite increase in number and size of the blood vessels.

The submucosa shows a round cell infiltration of only the more superficial layers (see Fig. 31) except in localized ulcerated areas, where erosion of bone is seen with many osteoclasts at the margin. With lack of free drainage of the purulent exudate this bone erosion may become more widespread to involve larger areas in the tract. Associated with this chronic suppuration there is marked sclerosis of the surrounding bone which begins at the periphery and advances toward the antrum, with a resulting eburnated mastoid process in long-standing cases.

Cholesteatoma.—The presence of stratified squamous epithelium lining the middle ear cavities in long-continued suppuration has been frequently observed. Nager reported an incidence of one case of cholesteatoma to three of chronic middle ear suppuration, from statistics taken over a twenty-year period. A complete discussion of the etiology and pathogenesis does not fall within the scope of this short article. The histopathology of cholesteatoma and a brief outline of the various concepts of its development follow.

The microscopic findings in cholesteatoma are quite characteristic. The lumen is filled with irregular sheets of lamellated amorphous material which are the desquamated surface layers of the epidermis. These are arranged in a concentric fashion and usually contain exudate in the center. The lining consists of an epithelial layer of typical stratified squamous epithelium with a thin submu-

cosa. As in other locations, the subepithelial layer consists chiefly of connective tissue fibers, some of which are elastic. and many blood vessels. The bony surface shows marked evidence of pressure atrophy, as well as absorption around the neighboring haversian and Volkmann's canals, as was seen in areas of affected bone in acute suppurative process. It differs, however, from the latter process in that the bone erosion is usually much more extensive, with the occasional involvement of the labyrinth capsule as well as other surrounding structures. Here, as in the other chronic suppurations, dense sclerotic bone is found surrounding the affected area.

There are numerous concepts regarding the development of stratified epithelial lining in the air spaces.

True or Primary Cholesteatoma.—Observers for many years have felt that aural cholesteatoma rarely may develop from misplaced embryological epithelial rests. McKenzie, in a recent study, suggests the possibility that all cases of cholesteatoma may be primary with the associated chronic infection as a secondary manifestation, but he admits the inability to explain the frequent occurrence of these tumors.

Pseudo or Secondary Cholesteatoma.—Metaplasia of the mucosa is thought to result from long-continued chronic irritation or from desiccation of the lining cells due to large perforations in the drum. Wingrave feels that this process is similar to the metaplasia of mucosa observed in the nose and other cavities where there is abnormal aeration.

Ingrowth of epithelium from the external ear is commonly observed. This may occur through large or marginal perforations following acute necrotic otitis accompanying scarlet fever. The mucosal lining has been largely destroyed and is replaced by immigration of the cutis layer of the drum or external canal wall. When the tympanum only is involved there is adequate exit for the purulent exudate and exfoliations from the surface layers of the epithelial lining. However, if the attic and antrum are affected, the tendency for occlusion and pressure phenomena is great.

Cholesteatoma may also frequently develop during the course of a chronic suppurative otitis, as the result of the valve-like action of the swollen or hyperplastic membranes separating the tympanum from the attic and antrum. The partial vacuum thus produced causes a retraction of Schrapnell's membrane. With the occurrence of adhesions in this region the pars flaccida may become so retracted

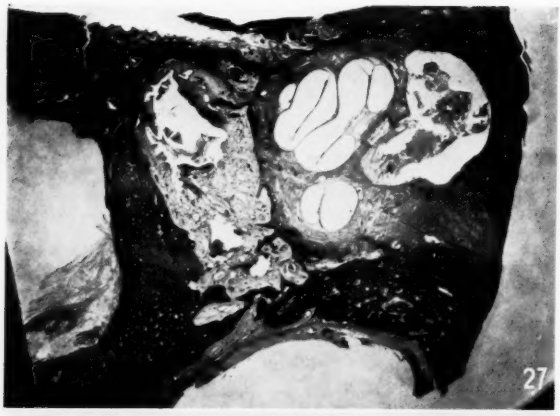

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Fig. 27. Photomicrograph: Vertical Section through Tympanum of Adult. Acute suppurative otitis media and mastoiditis with lateral sinus thrombosis, superimposed upon a long-standing chronic otorrhea.

Fig. 28. Photomicrograph: Horizontal Section through Tympanum of Infant with Chronic Otorrhea.

Fig. 29. Photomicrograph: Polypoid Mass of Granulation Tissue Near Drum Membrane (Medium High Power).

Fig. 30. Photomicrograph: Lining Mucosa (High Power). Taken from aditus of ten-year-old child with history of chronic suppurative otitis media for seven years. *c*, columnar ciliated epithelium.

Fig. 31. Photomicrograph: Lining Mucosa in Chronic Otorrhea.

as practically to line that portion of the attic. Either with or without perforation the invagination of the squamous epithelium may proceed further into the attic and antrum with very poor possibilities for adequate drainage.

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XVIII

HERPES ZOSTER OTICUS WITH FACIAL PARALYSIS AND ACOUSTIC SYMPTOMS: A SUBJECTIVE EXPERIENCE

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CLEVELAND

This syndrome of zona was first elaborated by J. Ramsey Hunt,[1] a neurologist, and was initially presented by him in 1906 before the American Neurological Association. However, the otolaryngologists have largely dispossessed the neurologists of their interest in this entity, probably through squatter's rights.

In the thirty-five years since Hunt crystallized medical thought pertaining to posterior poliomyelitis, as it is sometimes called, relatively few cases have been reported in the literature and surprisingly little has been added to or subtracted from Hunt's original concepts of the etiology, anatomy and pathology of this disease. For this reason one may briefly and categorically make the following statements:

Up to the time of Ramsey Hunt's publication, the only recognized localization of an herpetic inflammation of a cranial nerve ganglion was the gasserian, and all cephalic herpetic symptoms were attributed to this source.

For this belief, Hunt substituted the present one of considering herpes zoster of the face and neck as due to an involvement of ganglia containing the posterior spinal or unipolar type of cell as found, for example, in the spinal ganglia of the posterior roots as well as in the gasserian and the geniculate ganglia.

The histopathology of this ganglionic involvement was shown by Head and Campbell[2] to consist of well marked edema, round cell infiltration, cellular degeneration and hemorrhagic change. As the inflammation subsides, there is found connective tissue proliferation, in some cases to the point of scar formation. This inflammation also extends along the associated nerve trunks and has been known to involve the associated brain substance in certain instances.

The exact cause of this condition is unknown, but it is considered to be a specific virus infection with definite serum reactions and

antibody production.[3] Exposure to cold and heat as well as exhaustion, shock and local infection are mentioned as possible predisposing factors.

The zoster zone for the geniculate ganglion comprises, roughly, the drum surface, the external auditory canal and much of the anterior surface of the auricle. This is bounded anteriorly by the gasserian zone and posteriorly by the cervical zone, but none of these zones has exact boundaries, there being some overlapping.

The geniculate ganglion, though situated on a motor nerve, is sensory in type, having unipolar cells, and consequently is the homologue of the spinal and the gasserian ganglia. With its afferent fibres, the pars intermedia of Wrisberg, and its efferent fibres, the petrosal nerves, it forms the sensory system of the facial nerve. Hunt,[4] however, points out that phylogenetically this system is a recessive feature.

To understand the varied symptoms of zoster of the head and neck, it is necessary to realize that herpes in any zone not only signifies an infection of the particular ganglion supplying that zone but also a possible spread up and down in a sequential fashion involving neighboring nerves and ganglia, although to a less extent. An example would be geniculate zoster causing pain or herpes in the throat.

Ramsey Hunt[5] divided geniculate zoster symptoms into four main groups depending upon the severity and spread of the infection. Gradated from the least to the most severe, these are: simple herpes of the external ear; simple herpes plus facial palsy; simple herpes, plus facial palsy, plus mild disturbance of the acoustic apparatus, such as diminished hearing with or without tinnitus; and last, a severe infection characterized by simple herpes, plus facial palsy, plus a Ménière-like complex such as deafness, tinnitus, vertigo, nystagmus and nausea. Of course, various combinations of these four groups are possibly more often the rule than the exception.

The treatment is empirical and symptomatic. It includes leeching the mastoid area, removal of foci of infection, high frequency current locally, cocaine topically to the sphenopalatine ganglion, foreign protein injections, convalescent serum and roentgen therapy (Hill[6]). To these I would add pitressin, thiamin chloride and nicotinic acid.

I desire to report briefly my own personal subjective experience with this disease, of which I was a victim. My curiosity about this

malady, of which I had never seen a good example, was rewarded by my serving as my own private, though unwilling, guinea pig.

November 18, 1939.—The day cold and windy. Attended a colleague's funeral and sat on a cold, open veranda with my hat off during the service. Drove several miles with car window down. Very conscious of cold wind on left side of head and in left ear. General health good. Feeling fine. Habits good. No personal history of neuralgic disorders of any kind. Blood Wasserman negative. Teeth and sinuses negative for infection. Tonsils out. Otitis media in childhood. Tympanic membranes intact. Hearing grossly good in both ears with the right slightly the better. Premonitory aura, none.

Hereafter the succeeding days will be designated as so many days following this exposure.

1st day.—Feeling well except for the beginning of what I thought was a furuncle in the concha of the left ear between the tragus and antitragus. This area gradually became somewhat swollen and more uncomfortable with the discomfort extending toward the mastoid tip. Though I was not then aware of it, this was the typical herpetic eruption appearing within the zoster bone of the geniculate irritation. In 1871, Dr. Anstie[7] in England acquired the dubious distinction of being the first and until now the only physician-patient to record his subjective experience with zoster oticus. He also misconstrued his initial symptoms, thinking that he was developing an abscessed ear following exposure to cold. Happily for Dr. Anstie, his symptoms consisted entirely of herpes and otalgia, and he recovered completely in three weeks.

3rd day.—Supposed furuncle more bothersome. Awoke during the night, which was usual, but slobbered some water on attempting to drink, which was unusual. Disturbed by what I considered my clumsiness. This was the first evidence of facial weakness (which, of course, I did not recognize) affecting the orbicularis oris and preventing proper approximation of lips to glass.

4th day.—Symptoms now in profusion. While eating lunch, the left side of lips felt swollen and awkward and somewhat hypesthetic. While using the head mirror much bothered by photophobia and tearing of the left eye. In the evening, first noticed asymmetry of the mouth which was beginning to pull to the right. A big surprise came when I was cleaning my teeth before retiring—expectorating was such a distorted and perverted movement as to be

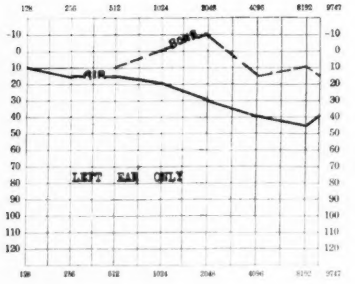

Chart 1. Audiogram of left ear using masking device for bone conduction showing hearing 31 months before attack.

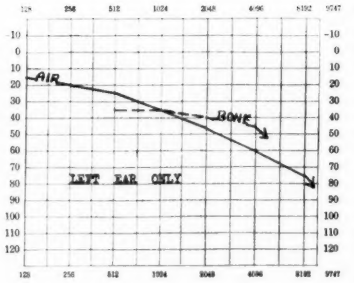

Chart 2. Audiogram of left ear using masking device for bone conduction showing hearing about 1½ months after attack.

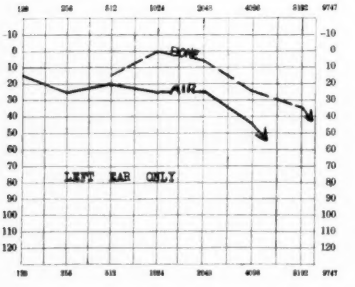

Chart 3. Audiogram of left ear using masking device for bone conduction showing hearing about 2½ months after attack.

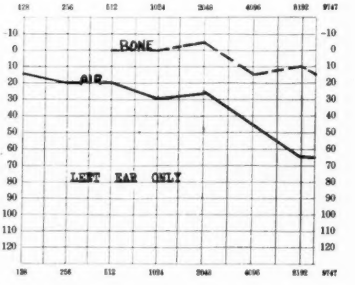

Chart 4. Audiogram of left ear using masking device for bone conduction showing hearing 13 months after attack.

actually funny, provided one could see the joke. Conscious of beginning hyperesthesia of scalp extending from the temporal area upward and backward to the vertex and the occiput. Here was early evidence of Ramsey Hunt's hypothesis of the extension of the inflammatory process from one ganglion and nerve to another.

5th day.—Closed left eye with difficulty. Much annoyance from epiphora and photophobia.

6th day.—First evidence of severe otalgia. Lancinating pains of extreme severity from deep in left ear to the temporal and the mastoid areas. Left ear began to feel very stuffy and blocked although the canal was quite patent. Hearing in left ear definitely diminished. No vertigo or nystagmus or tinnitus. Unable to read because of excessive lacrimation and photophobia. Complete paralysis of all branches of left facial nerve. Left eye glassy and staring. Much drooling on attempting to drink. Food, if chewed on the left side of the mouth, quickly lodged between the cheek and teeth and had to be dislodged by pressure on the outside of the cheek. Consultation with a neurologist, Dr. Karnosh, and an otolaryngologist, Dr. Kinney, proved my auricular furuncles to be herpes and provided me with the correct and complete diagnosis. Left auricle and preauricular area slightly swollen. Began taking thiamin chloride and nicotinic acid as well as using infra-red heat locally.

7th day.—Otalgia very severe—worse at night and very little affected by anodyne and sedative. Some fibrillary twitching of muscles about ear and left side of chin. Slight left-sided headache.

8th day.—Hearing worse—unable to hear watch tick in left ear, whereas had heard it at three inches (3/18). Ear feels very full with lateralization of my spoken voice to the affected ear. Began daily injections of pituitrin, which is followed in a few minutes by a roaring tinnitus lasting about one hour. Temperature 99.2 by mouth. Fleeting vertigo.

9th day.—More vertigo with some tendency to fall to the left. No tinnitus except for slight intermittent pulsating fullness in left ear synchronous with the heart beat.

10th day.—Vertigo worse with nausea but no vomiting. Considerable anorexia. Eating difficult because of tendency to bite the buccal surface of left cheek accidentally. Not aware of any gustatory disturbance of left side of tongue and able to demonstrate only a slight diminution of taste sense by testing.

11th day.—Vertigo continues. Some nystagmus to the right only when head is tipped backward. Marked hyperacusis and odynacusis to all sounds including my own voice and especially piano music and the radio. There is no Rhomberg and no past pointing. As Perlman[8] suggests, it is likely that the hyperacusis experienced here was due to paralysis of the stapedius muscle and was of the degree which he terms "hyperesthesia dolorosa".

14th day.—Otalgia severe and can be provoked by turning head to the right or by chewing, swallowing or talking. The otalgia now has a peculiar and characteristic "Roman candle-like" progression from within the depths of the temporal bone along the canal to the auricle where it seems to explode with a veritable shower of punctate stabbing shocks all over the auricle and adjacent temporal and mastoid areas. The marked hyperesthesia of the scalp is lessening. There is pain on deep pressure over the temporal, maxillary and mandibular areas. This is especially true of the region of the canine fossa. Sensory changes in the trigeminal zone in severe geniculate zoster are accurately commented upon by Ramsay Hunt.

17th day.—Otalgia frequently starts now by swallowing. Pain begins at the level of the upper border of the thyroid cartilage and extends deeply into the ear and thence externally. Much spasmodic twitching of the left side of face including forehead, eye, nose, mouth and chin. The most annoying is the twitching about the left eye. There is collapse of the left ala and resulting asymmetry of the anterior nares as well as nasal blockage on the left side. Here is probable evidence of beginning recovery of the facial nerve, possibly largely through lessening of edema within the facial canal. Nasal blockage on the left possibly due to collapse of the left ala plus the congestion incident to excessive lacrimation together with some probable irritation in the sphenopalatine ganglion.

20th day.—Nocturnal otalgia increased by cold air in sleeping room, so now sleep with head encased in surgeon's head gear, which makes a good night-cap. Otalgia lessening and hearing improving slightly. Vertigo still bothers. Now substituting pitressin for pituitrin.

22nd day.—Left ear very stuffy with sensation of a wooden plug having been driven deeply into the canal. Tinnitus marked. For the first time noted very slight voluntary movement of left side of face from mouth to temporal area. Valsalva inflation temporarily lessens stuffiness of the ear and improves hearing. The effectiveness

of the Valsalva technic would indicate the presence of tubal blockage which could have been due to closure of the eustachian tube owing to weakness of the tubal dilator muscles. However, there was no objective paralysis of the soft palate.

24th day.—This day displacusis was experienced for the first and only time. The occasion was the whistling of a member of the family.

27th day.—Diminished otalgia and vertigo. Possibly fifty per cent improvement in facial paralysis with least recovery in movement of left side of upper lip and nose. Beginning to notice involuntary activation of the left orbicularis oculi muscle when making certain chewing or nasal movements. This is an abnormal facial movement termed an "associated movement". Fowler[11] attributes this phenomenon to splitting of axons in the neuroma, which he states invariably forms at a point of severe nerve injury whether toxic or traumatic. It is supposed that there is a consequent enervation of several parts of the face by neurofibrils from a single axon.

29th day.—Starting today there was evident an intermittent fluttering sensation in left ear induced by any sound. This fluttering sensation seemed the subjective counterpart of the fibrillary muscular twitching present on the face. Vertigo now present only when I lie on my back or left side. It is likely that this fluttering represents a recovery response of the stapedius muscle and possibly of the tensor tympani through the otic ganglion.

32nd day.—Drove my car today for the first time. Greatly disturbed by the up and down motion of the car but not by side sway. This same sensation was present when walking but to a less extent. Facial paralysis rapidly disappearing. It would seem that this represents an irritability of the labyrinth especially of the otolithic apparatus.

42nd day.—Much annoyance from marked vertigo and nystagmus resulting from turning on my left side at night. This is severe enough to awaken me and can be prevented only by turning very slowly. The nystagmus appears to be chiefly horizontal judging from the dancing of the shadows on the wall. The vertigo and nystagmus have a latent period of possibly ten seconds and last about thirty seconds and then completely disappear even though I continue to lie on my left side.

After the lapse of a year, I notice the following residual sequelae:

1. A subjective sense of left facial weakness which objectively is not present at all or only to a slight degree.

2. Slight tenderness of the tissues in the left infraorbital and supraorbital areas.

3. Sensitiveness of left middle ear to cold and especially drafts of cold air.

4. Occasional deep lancinating pain in left ear, especially in the presence of an acute rhinitis.

5. Further impairment of hearing in left ear over that present at time of attack.

6. Periods of intermittent fibrillary fluttering in left ear aggravated by an acute rhinitis.

7. Occasional fleeting vertigo.

8. Involuntary associated movement of left orbicularis oculi muscle especially bothersome when eating.

SUMMARY

From the point of view of the physician-patient there are described the subjective symptoms of an attack of herpes zoster oticus associated with facial paralysis together with certain vestibular and auditory experiences.

So inclusive a train of symptoms is probably rare, but contrariwise, it is likely that many lesser attacks manifesting only otalgia or herpes of the ear, are mistakenly attributed to other causes.

10515 CARNEGIE AVE.

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Clinical Notes

XIX

(From a Group of Case Records Collected and Abstracted Under the Title of "My Most Interesting Case," By Noah Fabricant, M.D., Chicago)

BILATERAL JUGULAR LIGATION WITH RECOVERY

J. Mackenzie Brown, M.D.

Los Angeles

The infrequency of bilateral jugular ligation places this among my most interesting cases.

J. G., a male, aged five, suffered second and third degree burns on March 15, 1930. Measles developed during hospitalization. One week later bilateral bronchopneumonia set in and the following week the left ear began to discharge spontaneously. He was transferred to the Los Angeles Children's Hospital, where I first saw him on April 9, 1930. The left ear canal was full of pus, the membrana tympani was thick and red, and there was a posterior perforation. The mastoid was tender over the region of the antrum. Culture from the left ear showed hemolytic streptococci. The right membrana tympani was normal in appearance and function. The tonsils were hypertrophied and reddened, and there was posterior cervical adenopathy. The temperature was 103 degrees, pulse 125, and respiration 40. The blood count showed hemoglobin 75 per cent, R. B. C. 3.5 million, and W. B. C. 14,000.

On April 12th, the right membrana tympani was incised. The patient remained febrile, and repeated blood transfusions were given. On April 14th marked symptoms of left mastoiditis were present, and the following day a left mastoidectomy was performed. Culture from the mastoid showed hemolytic streptococci. The temperature continued to fluctuate from 100 degrees to 104 degrees, and the otorrhea from the right ear became more profuse. Definite signs of mastoiditis developed in the right mastoid, and this was

operated upon April 21st. The culture also showed hemolytic streptococci.

Following these operations the patient still remained febrile. Free straw-colored fluid was withdrawn from a painful right knee joint on April 25th. Transfusions were given when indicated. Both ear canals remained dry, the perforations healed, and there was very little drainage from the post-auricular wounds. Repeated blood cultures were negative.

The persistence of a septic temperature in the absence of any other findings prompted me to re-explore the mastoids on May 6th, at which time both sinuses were uncovered. A few granulations were found on the left sigmoid and not disturbed. The septic temperature persisted, and on May 8th the patient had a chill. On May 9th, the left sigmoid sinus was opened and found to be thickened in the area where the granulations were seen at the previous operation. No thrombus was found, and the sinus was packed. The left internal jugular vein was then ligated above the facial vein. The daily high temperature subsided, 101 degrees being the highest point until May 19th when a chill occurred and the temperature rose to 105 degrees plus. Positive blood cultures were obtained on May 21st and 23rd.

On May 24th the right sigmoid sinus was investigated. There was only a small area of thickened wall at the lower part, but no thrombus was found. The sinus was packed, and the right jugular vein was ligated above the facial. The temperature dropped to 102 degrees on the following day and continued to decrease until it became normal June 1st. There were no more chills. Blood cultures were negative from then on. He was discharged from the hospital on July 2nd.

The temporal, facial and external jugular veins were moderately dilated for a couple of weeks, following the tying of the second jugular vein, and then gradually returned to normal. There was also an increased tortuosity of the retinal veins.

The patient, now fourteen years old, is mentally and physically well, and leads a normal life in every respect.

My main purpose in presenting this report is to demonstrate a bilateral jugular ligation, without serious resulting circulatory disturbance. In this case the pathologic process in the sigmoids was phlebitic and not thrombotic. When there is a definite indication for ligation, the surgeon should not hesitate to ligate both internal

jugular veins and should always spare the facial veins. A reasonable time should elapse between the tying of the two veins in order to allow for development of compensatory circulation. The venous channels from the cranial cavity were spared a heavy load by compensatory dilatation of the temporal, external jugular, and facial veins.

1136 W. Sixth Street.

MASTOIDITIS AND MENINGITIS DUE TO PNEUMOCOCCUS TYPE I

LeRoy A. Schall, M.D.

Boston

H. P., aged 12, developed acute otitis media with mastoiditis and meningitis due to pneumococcus Type I.

A simple mastoidectomy was performed, with removal of the tegmen, linear incision of the dura followed by repeated lumbar punctures, and intraspinous and intravenous use of pneumococcus Type I serum supplemented by human complement. Recovery took place.

One year later the patient had a sudden memory aphasia followed by convulsions and unconsciousness. The only neurological sign was a questionable left facial weakness. Lumbar puncture showed fluid under increased pressure; the cell count was normal. I felt sure that he had a left tempero-sphenoidal abscess, but at operation none was found. The patient recovered.

Since then the patient has had three such episodes. During these occasions he presented the symptoms of a brain abscess, loss of consciousness, loss of reflexes on the right side, increased pressure on lumbar puncture, and a fall in the respiratory rate.

He now carries a letter stating that if he is found during the course of one of these attacks recovery will usually occur within six to twelve hours. This letter has saved him from several brain explorations.

243 Charles Street.

BILATERAL GRADENIGO SYNDROME WITH RECOVERY

RALPH A. FENTON, M.D.

PORTLAND, OREGON

An undernourished, undersized, anxious boy of seven was brought to us about ten years ago with well-marked bilateral mastoiditis. The family physician had permitted both ears to "break" and had attempted to manage the case for three weeks until the temperature reached 102 degrees. Both mastoids were cleaned out thoroughly, and the child left the hospital on the fifth day, although he required dressings at home for three weeks because of continuing headaches and fluctuations in temperature. Because his white count remained about 16,000 he re-entered the hospital for whole blood transfusions. There was, incidentally, a good deal of pain in the right lower jaw, and three suppurating teeth were removed. Little improvement in his general condition took place. Exuberant granulations were removed from both mastoid wounds, which continued to discharge slightly for a second month. Low resistance was evidenced by a high lymphocyte percentage.

Although radiographs showed that all cells, including fairly large zygomatic extensions, had been removed at the original operation, we decided that following another transfusion both mastoids should be cleaned out. When this was done little evidence of clean healing was evident. Temperature remained high, with the white count nearly 18,000, and headache was persistent.

Two days later the right external rectus was found to be paralyzed, with distinct weakness of the left external rectus, increasing in 24 hours to complete bilateral sixth nerve palsy. Some increase in discharge from both ears was noted; thick yellow pus contained a predominance of hemolytic staphylococci. Radiographs in the Rhese and Stenvers projections showed dark, fairly large cells running to the tip of both petrous bones.

Five days later, since no improvement occurred, the roots of both zygomatic processes were taken out, the dura exposed above the attic, and flat rubber strip drains placed along the petrous roofs for about 2 cm. No evidence of epidural pus was detected on either side.

Convalescence was stormy, and the patient left the hospital after another month. Granulations had to be curetted from the right

mastoid wound, still open, some three months later. Healing, however, was not complete for another month—this ten months after the original mastoid involvement. Marked hyperopia and convergent strabismus required glasses; both sixth nerves were still out.

Two years later the left eye was normal, and the ears were dry at the time the tonsils and adenoids were removed. Four years later both eyes were straight and the sixth nerves normal. He still requires a hyperopic correction, and when he is tired, the right abducens is a trifle weak. Hearing is normal.

Veteran of several hospitals, this husky youth of seventeen seems to Doctors Lupton, Bolton and me an exceptionally interesting case.

1020 S. W. Taylor Street.

THROMBOSIS OF THE CAVERNOUS SINUS, WITH BILATERAL BLINDNESS CURED BY AN UNORTHODOX PROCEDURE

Samuel J. Kopetzky, M.D.

New York

Every surgeon is occasionally confronted with a situation where his skill and resources are taxed to the utmost. For the most part his efforts in the face of a losing fight against a systemic infection and distressingly acute local manifestations are futile. The case which I shall here outline is therefore the more interesting, because in the face of a hopeless prognosis by all the consultants on the case, in the presence of total bilateral blindness, repeated chills, septic temperature, accompanied with its usual blood picture, the patient nevertheless made a complete recovery and regained her sight.

Upon a July evening as I was about to leave for a summer vacation, a phone call held me to see a young girl whose ear infection had been diagnosed as a septic sinus thrombosis. I went to the consultation and stayed with the patient until the Labor Day holiday in the following September.

The lateral sinus thrombosis was eradicated. The internal jugular vein resected and supporting systemic treatment instituted. Whole blood transfusions were administered. In spite of all therapy

there developed the unmistakable signs and symptoms of a cavernous sinus thrombosis. The usual eye picture was complicated in that the vitreous humor of the eyeballs of both eyes became opaque with purulency. It was impossible to observe the fundi.

The jugular bulb had been laid free at the operation for the eradication of the thrombosis in the lateral sinus and bulb and was dry. No plugs were necessary because no bleeding came from it. It was presumed that the thrombotic process had spread into the veins contributory to the jugular bulb and that the cavernous sinus had become involved through the superior petrosal.

Suggestions were advanced by me and others that the classical surgical procedures be undertaken upon the cavernous sinus, removing one eyeball, to gain access to the desired field of operation on the cavernous sinus. The parents of the patient denied us this operation, saying that they would rather see their only daughter dead than alive and deformed, with only one eye, particularly since the ophthalmologist could not predict that sight would be recovered even in that eye. I received permission, however, to do anything I thought would be helpful except to perform that kind of an operation. Realizing what I was up against, I made a plug to fit snugly and air-tight against the jugular bulb wall which it was designed to plug. Through this I passed a tightly fitted glass tube. I connected the glass tube with stiff rubber tubing, inserting a U-tube in the circuit to observe what it might collect. The tubing I connected with a strong turbine suction machine and set the suction machine in motion. No anesthesia was necessary. The child was unconscious. At first nothing happened, and then a long clot was drawn out from some tributary of the jugular bulb, followed by free blood. The wound in the jugular bulb was then plugged off with iodoform gauze to stop the bleeding, and the outcome awaited. Slowly but surely, the sepsis disappeared, and the patient recovered. The eyeballs cleared of their purulent opacities, and sight was restored.

Today, some years subsequently, there is still a small segment of optic atrophy in a segment between 11:30 to 1:30 on the fundi of both eyes, but the only untoward after-effect of the illness is the difficulty of seeing a segment of the floor. The patient has re-educated herself and protects herself against this handicap when walking.

I never reported this case in our scientific journals because I am not sure from where the recovered thrombus came. The original case history is long, covering as it does a daily record from the first

week of July to the first week of September. It illustrates that when faced with a desperate situation, occasionally surgical ingenuity and courage are rewarded. This happened in the days before chemotherapy.

71 E. 80TH STREET.

A CASE OF CHRONIC SUPPURATIVE OTITIS MEDIA, FACIAL PARALYSIS, CIRCUMSCRIBED MENINGITIS AND ACUTE LABYRINTHITIS

HOWARD C. BALLENGER, M.D.

CHICAGO

This case is of interest not because of each individual malady from which the patient suffered, but rather because of the unusual sequence of events with final recovery (before the advent of sulfanilamide). It is of further interest because the exact pathology is unknown and hence some speculation is permissible.

Miss G. H., a nurse, 27 years of age, was admitted to the Evanston Hospital January 1, 1935. She gave a history of having had a chronic suppurative otitis media of the right ear for about one year. Two weeks before admission she developed a nose cold and an increased ear discharge associated with pain over the right mastoid region. This was followed in a day or so by a persisting facial paralysis. Two days before admission she developed a stiff neck and severe headache. Both the headache and stiff neck were persistent and increased in severity. She had been running a slight temperature of one or two degrees throughout the present illness.

Examination revealed a rather slight young woman, evidently quite ill, complaining of increasingly severe headache, stiff neck, discharging right ear and inability to move the right facial muscles. Her mouth temperature on admission was 102 degrees F. From the right ear there was a profuse purulent discharge. Tenderness was present over the right mastoid region. A right facial paralysis was present. The neck was somewhat rigid. The eyes, pupils and reflexes were negative. Spinal fluid was turbid, under slight pressure, and showed a cell count of 1,130. Organisms were not demon-

strated. The Ross-Jones' test was negative, and the Pandy test showed a trace of globulin. The leucocyte count was 12,700 and the red cell count 4,270,000. The urine was negative except for 10 to 15 pus cells to the high power field. Twelve hours after admission nausea and vertigo developed. Spontaneous nystagmus was present upon looking to the right, this being the affected ear, and also upon looking upward.

The morning after admission a radical mastoid operation was performed in which the dural plate of bone was removed (the size of a quarter) over the middle ear and mastoid cavities. A cigarette drain was inserted and the mastoid wound partially sutured. On the third post-operative day she complained of difficulty in focusing the eyes. Objects appeared to be upside down. This seemed to clear after a few days. Following the operation the severe headache, vertigo and nystagmus persisted for about two weeks and then gradually subsided. The patient was discharged from the hospital on the 21st post-operative day. Following the release from the hospital slight nystagmus, induced by looking to the right or looking up, persisted for about two more weeks. Hearing in the affected ear showed marked diminution to the spoken voice. The facial paralysis persisted for two months after the operation, at which time the patient left the city and further contact was impossible. All discharge ceased from the ear and mastoid cavity within six weeks after the operation.

Naturally the question arises as to the sequence of events. It is improbable that the usual sequence of a chronic suppurative otitis media with an acute exacerbation extending to the labyrinth and then to the dura occurred. Rather it seems more probable that the otitis extended to the dura first, and then to the labyrinth by way of the internal auditory canal or by way of the blood vessels. It is also quite possible that the labyrinth was not involved by direct extension of the infection either from the middle ear or from the meninges, but that a subdural abscess or some other infective process produced the labyrinthine symptoms from a direct involvement of the eighth or other cranial nerves. I am inclined to this latter view inasmuch as recovery occurred rapidly after the dural drainage, and the mastoid and middle ear cavities healed and were dry within the normal length of time.

25 E. WASHINGTON.

XX

A CASE OF LABYRINTHITIS AND MENINGITIS WITH RECOVERY

WILLIAM E. GROVE, M.D.
AND
GERHARD D. STRAUS, M.D.

MEMPHIS

Sulfanilamide has proved to be a boon in the treatment of otogenic meningitis, and the literature is full of reports of its successful use in this complication of aural suppuration. In cases of labyrinthogenic meningitis most surgeons have combined labyrinthectomy with the use of chemotherapy. We are reporting a case of labyrinthogenic meningitis cured by a radical mastoidectomy and massive doses of sulfanilamide without labyrinthine surgery.

REPORT OF A CASE

A seven year old white female entered Milwaukee County Hospital on Nov. 22, 1939 with a history of pain in the left ear of one week's duration, following a cold. During the three days before admission, the child had vomited several times. The night before admission the temperature was 105° F.

The past medical history was irrelevant except for a tonsillectomy and adenoidectomy one year prior.

The temperature was 102° R on admission. The left tympanic membrane was reddened and bulging. A small amount of discharge was present in the external auditory canal. The white blood count was 10,850 and hemoglobin was 75%. Physical examination was otherwise negative.

Because of the severe pain and poor drainage a myringotomy was performed and hot compresses were applied to the ear. The temperature remained somewhat septic until Nov. 26, 1939 when it rose to 104.6° R, and the patient complained of severe frontal headache.

Examination at this time revealed a horizontal rotatory nystagmus of Grade I on turning the eyes to the right or left, but this was definitely more marked on looking to the left. The Weber was referred to the left. The child was unable to hear a shout with the left ear, the right ear being masked with a Barany noise maker. No other neurological signs were present.

Lumbar pucture revealed a cloudy spinal fluid; there were 3,900 white cells present. Globulins were 4 plus and the sugar content was 26.6 mg. per cent.

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*From the Departments of Otolaryngology, Marquette University School of Medicine and Milwaukee County Hospital.

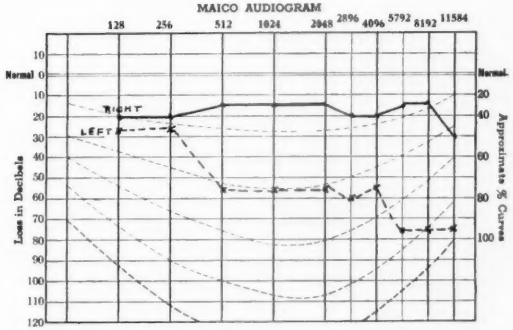


Fig. 1.

A smear and culture showed gram positive cocci in short chains. The blood count at this time was as follows: Hemoglobin 71%; R.B.C. 4,220,000; W.B.C. 23,700; neutrophiles 89%; lymphocytes 6%; monocytes 5%.

A left radical mastoidectomy was performed immediately. The mastoid cells were hemorrhagic, and no pus was present either in them or in the mastoid antrum. The middle ear was full of granulations.

The dura of the middle and posterior fossae was exposed widely but no gross pathology was noted. No labyrinthine necrosis was seen. The labyrinth was left intact, and the wound was packed with iodoform gauze. No plastic operation was done.

Immediately postoperatively 50 gr. of sulfanilamide was given subcutaneously in 500 cc. of normal saline. Four hours later this was repeated. Six hours later the patient was started on sulfanilamide and sodium bicarbonate aa gr. X every four hours, orally.

One hundred and forty gr. of sulfanilamide were given in the first 24 hours. This was decreased to 60 gr. daily for the next four days then to 40 gr. daily for 18 days. The sulfanilamide concentration in the blood was 23.8 mg. per cent after the first 24 hours, and was maintained at from 12-16 mg. per cent thereafter.

The Hg. and R.B.C. were low on two occasions but were bolstered by two transfusions of citrated blood. The spinal fluid cell count dropped from 3900 to 1,475 in two days and in five days to 400, when for the first time the spinal fluid was clear of streptococci and the spinal fluid rose to normal. By the eighth postoperative day the cell count was 31 and then gradually returned to normal.

Clinically the postoperative course was stormy for about one week; from then on however convalescence was uneventful. Physical examination on June 17, 1940 revealed the postauricular wound healed. A small amount of mucoid discharge was present in the middle ear.

Functional examination of the ears was as follows: Whispered voice: left ear, 2 inches, right ear, 20 feet; Spoken voice: left ear, 12 feet, right ear, 60 feet; the Weber lateralized to the left; the Rinne—left, air 55 seconds, bone 50 seconds; right, air 90 seconds, bone 45 seconds. Figure 1 shows the audiogram taken at this time.

A caloric test performed with 5 cc. of water at 80° F. was as follows: Left ear—latent period, 25 seconds, duration of nystagmus, 60 seconds; right ear—latent period, 15 seconds, duration, 80 seconds.

This case is presented because of two unusual features. First, it is a case of labyrinthogenic meningitis with recovery without labyrinthectomy. Second, despite the apparent bacterial invasion of the leptomeninges via the inner ear, the patient was left with a functioning labyrinth.

We wish to leave one thought. Since the use of the new chemotherapeutic agents has revolutionized the treatment and prognosis of otitic complications, perhaps in cases of labyrinthitis and meningitis without obvious necrosis of the labyrinthine capsule it may be advisable to procrastinate labyrinthine surgery until chemotherapy has been given a trial.

MARQUETTE UNIV. SCHOOL OF MEDICINE.

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, April 1, 1940

THE PRESIDENT, DR. GEORGE T. JORDAN, IN THE CHAIR

Diagnosis and Pathology of the Salivary Glands

R. BRUCE MALCOLM

(Abstract)

The essayist stated that the pathology of the salivary glands was unique. His illustrated talk was based on 167 cases seen during the past seven years, on each of which he had operated and from which he had seen each gross specimen and examined it histologically. The cases were grouped as follows:

Parotid:

Acute suppurative parotitis	8
Incised wound with VII paralysis	1
Papillary cystadenoma	3
Tuberculosis in parotid	1
Hodgkins' disease in parotid	1
Lithiasis with chronic inflammation	3
Mixed tumors	45
Carcinoma	23

Submaxillary:

Chronic obstruction of ducts	2
Lithiasis	5
Aberrant salivary in thyroglossal duct cyst	1
Cysts	3
Mixed tumors	6
Carcinoma	2
Resection for carcinoma of lip	55
Resection for malignant carcinoma	3
	77

Sublingual:

Cysts	3
Lithiasis	2
	5

There was a total of 167 cases.

Acute suppurative parotitis, while not common, is extremely painful and must be drained. It may be caused by ordinary lithiasis. The case listed with seventh nerve paralysis was due to a knife wound; the nerves were sutured with a satisfactory result. Papillary cystadenoma usually occurs as a small tumor of fairly rapid growth, not nodular, and may undergo malignant change. One case diagnosed as such proved to be tuberculosis of the gland, while another was due to Hodgkin's disease.

Mixed tumors are one of the great bugbears of surgery. At first there is simple hyperplasia, then fibroblastic hyperplasia; then they become anaplastic and one may find chondroblasts and even osteoblasts. They usually appear in the lower pole as a small, gradually growing nodule. Sudden enlargement may take place after a number of years, and then they must be considered malignant. One case cited was that of a young man who had a tumor mass the size of a walnut for years, which suddenly increased in size. Prior to that time he had had 20 X-ray treatments. Recently facial paralysis had occurred, with intense pain. When this triad, pain, rapid growth and facial paralysis, is present the diagnosis of carcinoma is confirmed. Seven of 23 carcinomata developed in ordinary benign

mixed tumors. Probably one in four will undergo malignant change, particularly if there has been partial removal and X-ray therapy. Squamous cell carcinoma may arise from such a tumor, and six such cases are listed in this series. Ten were of the adenocarcinomatous type. In one case, that of a girl aged five, with a tumor of the parotid gland, the biopsy showed rapidly developing adenocarcinoma. An attempt was made to remove the whole tumor but it was adherent to and invaded the jaw. It was necessary to remove the jugular vein, tie off the external carotid and remove the entire gland.

Another patient, aged 23, had a tumor mass in the floor of the mouth. A surgeon attempted to remove it, but actually removed the duct itself and its nippled orifice, resulting in a recurrent obstruction. Eventually it was necessary to do a complete extirpation of the gland. In a girl aged two, it was necessary to cut the hyoid bone and remove a mixed tumor of the salivary gland. Carcinoma is rare in the submaxillary glands, although two have been found in this series. Metastases in the lymph nodes necessitates removal of the submaxillary gland. In five cases, there was secondary involvement spreading into the submaxillary gland, which seems to be resistant to squamous cell carcinoma, as in 55 excisions of carcinoma of the lip only three showed involvement of the salivary gland.

The most malignant tumor is malignant melanoma with almost complete destruction of the salivary gland, which occurred in three cases. Diagnosis of all these tumors is usually made by palpation on the outside and through the mouth, which gives more information than any other procedure.

Electrocoagulation

JOSEPH F. JAROS

(Abstract)

The colored motion picture presents the actual process of electrocoagulation within body cavities such as the larynx, rectum and vagina, taken at a distance of four feet from the patient through special light projection, showing, greatly enlarged, the contents only of the cavities—tonsil, hemorrhoids and uterine cervix—and suggesting means toward the elimination of the most common objections to electrocoagulation, i.e., operative and postoperative pain, hemorrhage and scarring.

The film presents for the first time:

1. A new principle for the elimination of hemorrhage after electrocoagulation, based on the first histo-mechanical principle of Thoma and the researches on capillaries by Elliot R. Clark.

2. The presence of an anarterial area of the tonsil based on the research of Fetterolf and on over 800 cases of tonsil coagulation by the essayist.

3. The regeneration of glands and of even ciliated epithelium of the nasal turbinates and of the uterine cervix following severe electrocoagulation.

4. A partial analgesia of three or four days following topical application of five per cent diothane solution.

DISCUSSION

DR. FRANK J. NOVAK: We must all admit that we have seen a demonstration of very beautiful colored photography in the depths of a cavity, which is not easy to obtain. I hesitate to renew the controversy between those who remove tonsils by surgery and those who dessicate them. The matter has been discussed many times, and I have not changed my opinion. I must say that after observing a number of these cases completed by the essayist and having examined them with a critical—perhaps hypercritical—eye, I found no evidence of any retained or remaining lymphoid tissue in the fossae; perfectly pliable pillars and no evidence after a long period of any recurrence or creeping in of adjacent lymphoid tissue into the tonsil fossa.

I am eager to hear Dr. Rukstinat speak on some phases which will be of interest to you, and I would like to yield whatever time I have left to him.

DR. GEORGE J. RUKSTINAT: A microscopic study of six portions of turbinate and two uterine cervices subjected to electrocoagulation by Dr. Jaros and later surgically excised, disclosed interesting late results in tissue regeneration. The turbinates had been treated from six weeks to one and one-half years prior to their excision. Each one was covered by epithelium two or more layers thick, in some places stratified squamous in nature, but for the most part cuboidal. In a few of the small depressions ciliated epithelium was found. Variability in the type of covering epithelium depends, in some measure, on the exposure of the turbinates to aeration. The deeper structures such as glands and blood vessels were, however,

uniformly well preserved. Their number and distribution were normal and suggested no prior mechanical or thermal treatment. Although the number of tissues studied was small, Dr. Jaros and I realized that a preliminary report of the favorable findings would stimulate further investigation.

DR. THOMAS C. GALLOWAY: I was much interested in this subject at one time, and about ten years ago I tried all technics of electrosurgery in about 100 cases and concluded it was of advantage only in a few special cases or those with flat fragments in an open fossa. In going back to one of the instrument houses which used to hold crowded clinics to teach how to take out tonsils by coagulation, I found the place almost deserted this year. I asked one of the salesmen about it and he said, "Coagulation ain't the panacea it once was—short wave is the stuff now."

In spite of what has been said, probably the time loss, total disability and pain is considerably greater in coagulation than it is with proper dissection. I have seen Dr. Jaros work, and there is no doubt he exercises as much skill and care in doing this work as one who does good surgical tonsillectomy. It is still my opinion, however, that in hundreds of patients I have seen whose tonsils were originally treated by electrocoagulation there were not six cases I could consider good clean tonsillectomies.

DR. ROBERT H. GOOD: I think surgical diathermy is one of the most wonderful assets I have seen in the last ten years, but I thought the matter of dissecting tonsils had been pushed overboard, so far as attempting to remove the whole tonsil with diathermy is concerned. We tried this years ago, and I could not get patients to come back ten or twenty times; many did not have it completed. Another objection was that I never could tell where the limit of the capsule was. If the diathermy went too deep there was danger of destruction of tissue. It is fortunate that it is a painful procedure, and I am sorry a new anesthesia has been found which makes it painless for four days following the procedure.

DR. AUSTIN A. HAYDEN: I agree fully with what Dr. Galloway has said about this procedure. My experience with electrocoagulation of the tonsils has been entirely that of an observer. Very briefly, the results have been terrible in the cases I have seen.

The procedure shown tonight, with pictures six months or six weeks after operation, seemed to show very clearly the absence of scar tissue and complete pliability and even elasticity of the tissue of the pillars. I was very glad to see these pictures and I marvel at

the ability of the doctor to make photographs inside the throat. They are the best I have seen. I commend his photography, but I doubt his surgery.

DR. JOSEPH C. BECK: Since Dr. Jaros referred to me as one who had discarded this method of treatment, I wish to reply that this is not entirely correct. I have distinctly stated many times that I employ electrocoagulation for remnants of tonsils or recurrences of tissue at the base of the tongue, which I or my associates left in the throat. Where recurrences and infection occur in such tissues I have felt it better to destroy the tissue by electrocoagulation rather than by dissecting these stumps, which were in most instances not actual tonsil tissue. I have never felt that I wanted to remove tonsils by the method of electrocoagulation in contradistinction to the usual surgical method.

It was interesting from a pathologic standpoint to see the section following electrocoagulation which left the epithelium not only intact but apparently reformed. The late Dr. Bloodgood showed the use of the electrocoagulating knife which incised the skin, and showed that primary union occurred after such incision. It must be a special type of current which permits primary union without stitch abscess or suppuration following electrosurgical incisions.

I hate to put my opinion against that of the pathologist, but one section shown tonight looked to be so poorly stained as to be atrophic. Just because there are a few glands left is no reason to say it is normal. I do think it was a pretty good effect of coagulation having destroyed the good staining tissue.

In conclusion I wish to say that when any method is brought up and the attempt is made to show real work, as was done tonight, we should be open-minded and say it is good. These pictures were good; the color photography, aside from the excess of high lights, was excellent. The pliability of tissues after operation was certainly shown in these pictures; there is no question about that. There is far more mobility and less scarification than is usually seen following ordinarily performed tonsillectomy.

MR. HOWARD A. CARTER: The Council of Physical Therapy feels that the regular surgical removal of tonsils is the method of choice. In the serious cardiac cases, in severe toxic goitre and in the advanced tuberculous patient, or where other complications would make it inadvisable to use a general anesthetic, electrocoagulation would be indicated.

DR. ELMER KENYON: Many years ago Dr. G. W. Hudson Makuen expressed much concern about the desirability of tonsillectomy because of the frequent resulting impairment of the tissues of the fauces and their action following operation. He said, "You are making it impossible for children to develop singing voices." I would like to say that there are many cases in which tonsillectomy is recommended which should not be submitted to this operation. Those are cases in which the palate is short, and if through tonsillectomy you shorten it still more, which is almost inevitable, you may have a nasal voice as a result.

I used to say years ago that we should have two methods of approaching the tonsil: one, removal by tonsillectomy extracapsularly, and the other, removal of the lymphatic tissue within the capsule. Operation within the capsule is not attended with deformity. My thought and experience has been to extirpate the tonsillar tissue within the capsule with biting forceps. If we could add cauterization as a final step to this procedure, such an intracapsular operation should be an excellent one.

DR. L. Z. FISHMAN: I should like to emphasize the remarks of Dr. Kenyon with respect to the need for preventing even the slightest disturbance in the motility of the posterior pillar. Not only are the structures of the soft palate concerned with resonance and articulate speech—and these represent only gross acts of the palate—but they are also great factors in the quality of the singing voice. The fact that tonsillectomy can cause a derangement in the voice by disturbing the elasticity and motility of the posterior pillar is particularly evident in an unusual specimen which I encountered in my dissections of larynges. In this larynx, the pharyngopalatine muscle (posterior pillar) was found to be a direct combination of a number of muscle fascicles of the vocal muscle (lateral crico-arytenoid muscle) itself. Obviously in this instance the posterior pillar was an integral part of the laryngeal adductors. Certainly the greatest care must be exercised in performing tonsillectomies on children who may become singers. In the case of vocalists this operation should be avoided to the greatest extent possible; any substitute for a complete surgical tonsillectomy is to be desired in such instances.

DR. WALTER THEOBALD: Some few years ago you will recall that this subject was presented before this Society, and I think at that time it was pretty well hashed over and closed. Some twelve cases were presented by Rush Medical Clinic and, in that group, not one showed a satisfactory result. That was the admission of the operators as well as the observers. Dr. Beck has proposed that these cases

of Dr. Jaros' be brought in for observation. This presentation was given primarily to demonstrate some very fine photography, knowing full well that we would have a stiff argument condemning this procedure. Dr. Novak and I thought the work was worthy of presentation because of the excellent color photography inside a cavity. If it is the wish that this report be brought up at some future time, I would suggest that a small group be detailed to examine some of Dr. Jaros' cases and report to the Society.

Dr. Joseph F. Jaros (closing): I wish to thank the Society for its kind reception of this presentation. Dr. Good called attention to the striking elasticity of the pillars following electrocoagulation. This lack of scarring is especially characteristic following the multi-stage method and is especially desirable, as Dr. Kenyon clearly brought out in his discussion, in the supratonsillar fossae and in the posterior pillars. This feature alone is worthy of further study.

Dr. Novak and Dr. Galloway have kindly corroborated the film demonstration of possible satisfactory results. This possibility can be more frequent as pain is more and more eliminated, since absence of pain ensures more thorough and more rapid work.

The absence of recurrence is interesting and although, as Dr. Galloway has stated, complete removal may be more difficult through electrocoagulation than by surgery, recurrence and regrowth have not been observed in the many cases re-examined over a ten-year period in more than 800 cases. Removal has been satisfactory in cases of angina pectoris and coronary thrombosis.

The suggestion that completed cases might be presented before a committee is well taken. Fully 24 cases, treated during the past ten years, have agreed to submit to inspection.

Hearing Aids—Tubes or Carbon?

Austin A. Hayden

(Author's Abstract)

At present both tube and carbon aids have definite advantages and disadvantages. Both are being improved constantly.

Indications for each must be formulated from the experience of otologists, physicists, and manufacturers, with the final decision being made by the patient.

Tube aids give more brilliant hearing improvement in conduction losses where nerve function is good. In severe perceptive loss results are often disappointing.

Purchase from a firm that manufactures both carbon and tube aids for air as well as bone conduction, allows choice between the two and makes exchange of one for the other easier.

Since tube aids are much more complex instruments, the need for an impartial evaluating agency such as the Council on Physical Therapy of the American Medical Association, to set and maintain requirements for acceptance of standards of construction and performance as well as of sales ethics and advertising, is even more necessary with the advent of tube aids. Financial rating of the manufacturers is very important.

Although the lure of radio tubes is great, patients should not discard carbon for tube aids without careful investigation.

Tube aids are as yet more expensive to manufacture, buy, keep up and service.

Whether tube aids will supplant carbons, or vice versa, or whether each will eventually occupy its own definite field, the actual improvements that are being made in construction by manufacturers and in the prescription of hearing aids by otologists practically assures good results in the vast majority of cases properly fitted with a well-made hearing aid.

Even better instruments and better hearing seem to be on the way.

DISCUSSION

MR. HOWARD A. CARTER: I will not take your time to discuss the old mechanical aids such as rubber tubes, ear trumpets and fans. The first slide shows a schematic diagram of the carbon granule transmitter hearing aid. It is of a similar circuit used by one-way telephone and operates as follows: The sound energy impinging on the diaphragm sets it in motion, which changes the pressure on the carbon granules thereby varying the resistance in the circuit. The energy from the battery is varied in the entire circuit which passes through to the receiver. Varying current in this circuit changes the magnetic flux in the permanent magnet of the receiver and hence sets its diaphragm in motion, which in turn sets up a sound disturbance in the vicinity of the deafened ear.

The vacuum tube aid is somewhat more complicated. In the vacuum tube aid the crystal microphone is used. This crystal is a miniature generator developing a feeble electric current which is amplified by three vacuum tubes. The energy for amplification comes from the A and B batteries. Finally, the energy which has been amplified is delivered to the receiver, which may be either a crystal or electromagnetic type.

DR. G. H. MUNDT: I think many people share my predicament. I never know when to prescribe a hearing aid. I have read and heard a great deal about it, but I have never been quite clear as to whether a hearing aid should be prescribed for the person who has lost a little hearing, or wait until he is entirely deaf; or whether a hearing aid should be prescribed, or lip reading recommended. I wish Dr. Hayden would give us a word on that.

DR. ROBERT H. GOOD: Would a wet battery produce less noise, and would the tubes last longer with a wet battery than with a dry battery? I know the wet battery is more smooth. In dry batteries air bubbles form and change the voltage, which causes the noise.

MR. HOWARD A. CARTER: By wet batteries I understand you to mean storage batteries. The answer is that there should be no significant difference. It is assumed that "static" means the internal noises in the hearing aid. I am not quite clear as to why wet batteries should be more smooth, as you say. I should think a dry battery made up of several cells would be as smooth, if not smoother, than a small storage battery. It is possible, of course, to charge a storage battery over and over again. The lasting qualities of the hearing aid should not be any different, whether a storage battery or a dry battery is used.

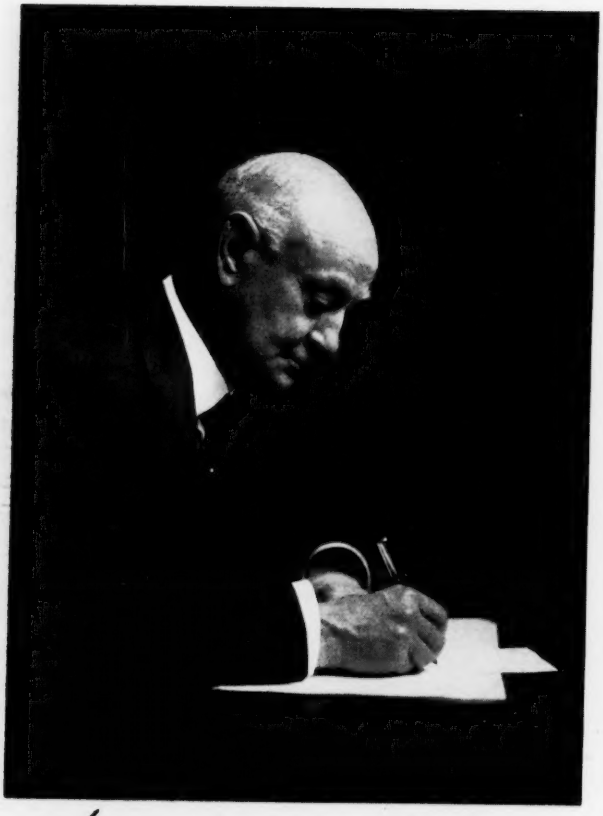
DR. L. J. HUGHES: I think the sooner a deaf person becomes adapted to a hearing device the better. It helps to retain the natural voice, and he does not develop the high nasal voice.

DR. AUSTIN A. HAYDEN (closing): The question that Dr. Mundt brings up is important. My answer would be to invite him to the Illinois State Medical Society meeting, where we are going to deal with this subject: "For whom should hearing aids be prescribed?" In general, they should be prescribed for people whose hearing is defective and can be improved by the aid. There are many exceptions to that—the psychologic factors, the matter of expense and the matter of pride. People do not want to wear these devices. As to lip reading, we regard it as a natural human hearing aid, and therefore lip reading and hearing aids should go together. Lip read-

ing is an art, an inexact art. It cannot be used unless strict attention is paid to the context of what is being spoken. Many sounds are formed in the same way by the lips, and the context determines what they mean.

In reply to Dr. Good's question about wet batteries (the small type operated with a few drops of fluid), that has been gone into extensively by the hearing aid companies. Their opinion and the opinion of patients has been unfavorable. Only one company pushes them, and, all in all, their sales have been very small. The battery expense of a hearing aid is $2.50 a month, which could be reduced considerably. It is astonishing that the only thing necessary is to use two batteries, alternating day by day, and instill two drops of water into the battery after it has been used. That simple direction is seldom followed. I have no evidence about whether they are harder on tubes than dry cells, or whether there is less extraneous noise.

With reference to Dr. Hughes' statement, the evidence is not at all conclusive. The natural voice depends upon the retention of bone conduction, and whether or not a hearing aid, even though worn as continuously as possible, will actually preserve the voice is something which is disputed. I know Dr. Hughes' experience and respect that very much, but there is much evidence to the contrary with regard to that opinion.



Yours sincerely
Herbert Hoover
Xmas. 1934

Elliott Brown

HERBERT TILLEY

1865-1941

The turn of the year marked the passing of one of England's grand old men of laryngology. Herbert Tilley, M.S., F.R.C.S., died on January 6th at his home, St. George's Hill, Weybridge, England. He was born in Somerset December 10, 1865.

Mr. Tilley was Honorable Consulting Surgeon (in the diseases of the ear, nose and throat) in University College Hospital, and Consulting Aural Surgeon in the Ministry of Pensions.

His long and fruitful career left a profound impression upon contemporary thought in English otolaryngology. He held many important posts in the medical world, and it was due largely to his initiative that the examinations for the diploma in ear, nose and throat surgery were established by the Royal College of Surgeons. He delivered the Semon Lecture before the University of London in 1934, on the subject, "Chronic Pyogenic Inflammation of the Antrum and Other Accessory Sinuses."

His volume, "Diseases of the Nose and Throat" (H. K. Lewis & Co.) is still one of the standard texts, and his contributions to the literature are to be found in many of the special journals of Great Britain, France and the United States.

He was at different times President of the Section of Laryngology and of Otology of the Royal Society of Medicine, President of the Section of Laryngology of the British Medical Association in 1910, and President of the Medical Society of London in 1931.

He visited this country on various occasions and was an Honorary Fellow of the American Laryngological Society and the American Academy of Ophthalmology and Otolaryngology.

During the First World War he was consulting surgeon to several military hospitals in and about London. For their services during this crisis, both he and Mrs. Tilley, who survives him, were cited by various charitable institutions.

Many an American colleague has received an hospitable welcome at the home of the Tilleys and will feel a personal loss at the passing of this genial friend.

A. W. P.

Abstracts of Current Articles

EAR

The Treatment of Chronic Deafness and Tinnitus Aurium with Prostigmine.

Houser, K. M., Campbell, E. H., and Schluederberg, H. (Philadelphia), J. A. M. A., 115:12:994, 1940.

The selected cases were treated with 1 cc. of prostigmine methylsulfate hypodermically, 1:2,000 dilution, every third day and 15 mg. of prostigmine bromide orally three times daily. Each case was treated at least two months, and audiometer tests were performed every two or three weeks.

Of fifty-six cases hearing was improved in none. There was questionable partial relief of tinnitus in seven.

DEAN, JR.

Removal of the Incus in Operations for Acute Purulent Mastoiditis; Indications and Results. (Indicazioni e risultati dell'asportazione dell'incudine nella operazione per oto-mastoidite purulenta acuta.)

Scalori, G. (Messina), Valsalva, 15:337-352 (August), 1939.

An accurate bibliographic research conducted by the author did not uncover a previous report on the auditory function in mastoidectomized patients in whom the incus was removed.

Twelve patients suffering of postinfluenzal, acute, purulent mastoiditis were selected for this experiment. In all cases the bone destruction involved the most anterior portion of the epitympanum. It is evident that the surgeon was obliged to demolish a considerable portion of all the antral walls, thus exposing completely the incudal fossa. The incus, being held in place by weak ligaments which often are wanting, may easily be displaced by the curets or the sponges during such an extensive surgical procedure.

Hearing tests were carefully taken in both ears at specific intervals, i. e., prior to operation, when the mastoidectomy wound was completely healed, and three months after dismissal. The author charts the hearing test of each patient in every detail in this paper. The voice, the watch and a series of tuning forks ranging from 64 d.v. to 4096 d.v. were used. For the Rinne, Weber and Schwabach

the 128 d.v. fork was employed. A uniform method of exciting the forks was carefully followed.

In eight of these patients the incus was removed to permit better drainage, where the lumen of the aditus was very narrow, and also fearing that the immobilized ossicle might necrose. In the eight patients whose incus was removed, the summary of the examinations revealed that 1) the postoperative course was undoubtedly greatly shortened; 2) a considerable loss of air conduction was disclosed by the cochlear functional tests; 3) there was a constant, annoying, high-pitched tinnitus.

In the four cases in whom the incus was left in situ, even though loosened or dislocated, 1) the hearing defect was very slightly diminished, if any; 2) no tinnitus was found, though in two of the cases there was a slight subjective noise due to cicatricial formations in the middle ear.

Scalori concludes that while the removal of the incus shortens the postoperative convalescence, it produces a marked hypacusia and an annoying tinnitus. Therefore, he definitely condemns its removal even though it may be displaced or necrosed. He maintains that this ossicle should be removed only when a radical mastoidectomy is to be considered, because the patient does not need any further care once the wound has healed.

SCIARRETTA.

The Portmann Operation for Blockage of the Endolymphatic Sac.

Waltner, Jules (Budapest), Rev. de Laryng., Otol., Rhin., 61:1 (Jan.), 1940.

This is a detailed description of the Portmann operation for vertigo. The surgical treatment of vertigo is reserved for extreme cases that have not responded to medical therapy.

From both the clinical and anatomicopathological point of view, blockage of the endolymphatic sac (Ménière's syndrome) is an actual nosologic unity.

The Portmann operation is based upon well established anatomical, physiological, and pathological facts and represents the only etiological operation for otitic vertigo. Excluded from this method of therapy are cases in which vertigo is precipitated by central lesions (bulbar and encephalitic), lesions of the cerebellopontile angle (tumors and arachnoiditides), labyrinthitis of the inflammatory type, and syphilis.

The Portmann operation presents the following advantages:

1. It is a simple operation with an easy route of access.

2. It is entirely aseptic and therefore not dangerous.

3. The operative complications (hemorrhage) are rare and of very little consequence.

4. The results are permanent. The attacks of vertigo are eliminated and in fifty per cent of the cases there is an improvement in audition. The operation does not result in a diminution or loss of hearing. However tinnitus is not as satisfactorily affected.

VIOLE.

NOSE

Incomplete Late Hemiplegia (Emiplegia incompleta tardiva per ferita da fuoco).

Jemmi, Carlo (Parma), Rivista Oto-Neuro-Oftalmologica, 16:6:436, 1939.

The author discusses a case of incomplete late hemiplegia in a patient who had received a rifle wound of the maxillary sinus, the zygomatic fossa, and the pre-stiloid fossa.

Afterwards he determines the place and nature of the cerebral lesion on the basis of anatomic and physiologic facts and symptomatology, and suggests the possibility of a fat embolism of the middle cerebral artery.

PHARYNX

Zambrini's Test Employed, Both Before and After Operation, on Patients Having Adenoids or Adenoids and Chronic Hyperplastic Tonsils. (La reazione di Zambrini negli adenoidei e negli adenoidei con tonsillite cronica ipertrofica prima dell'intervento e alcuni mesi dopo.)

DiBlasi, A. (Messina), Boll. delle malatt. Orecch., Gola, Naso, 57:295-306 (August), 1939.

Faust Zambrini believes that the humoral state of the individual becomes modified or altered during all pathological conditions; consequently these changes are also manifested in the saliva. The variations in the saliva are made evident by the ptyalin test of Zambrini which D'Arsonval calls the thermometer of vital resistance.

The ptyalin reaction is a chromometric chemical test produced by a special reagent mixed with the whole saliva. It has been used

extensively during the last few years both in France and Italy. Surgeons have employed it to determine the vital resistance prior to operation, and numerous clinicians in the field of medicine have adopted it for diagnostic, prognostic and prophylactic purposes. In the field of otolaryngology, DiBlasi is presenting this paper.

The materials needed for the test are the following: 1) A reagent composed of pure carmine of cochineal, gm. 1.00; trioxyanthraquinone, gm. 1.00; dioxyanthraquinone, gm. 7.00; rubia tinctorum, gm. 1.30; and 95% alcohol, gm. 1000. 2) A calorimetric chart beginning with a light yellow and gradually changing to a pale rose, ruby red and terminating with a deep violet. These colors are divided into sixteen parts. The light yellow is marked number one and the deep violet number sixteen. 3) A graduate test tube made of neutral glass and corked with a rubber stopper.

The sputum is collected on an empty stomach after the patient's mouth has been washed and cleansed thoroughly. Fifteen to twenty drops of the reagent are added to 1 cc. of saliva and shaken vigorously. The color acquired by the mixture is compared with the calorimetric scale in daylight and numerically recorded. The low numbers represent low organic resistance and the high numbers, high resistance. The presence of a foamy collar over the meniscus invariably indicates that the disease is caused by an infectious agent, more or less virulent, while its absence denotes lymphadenopathy. The clouding and consequently the sedimentation of the mixture must also be observed because the quantity and the rapidity of the sedimentation has a significance.

DiBlasi took a Zambrini test on fifty subjects averaging ten years of age, some with only adenoid hypertrophy and others with adenoid hypertrophy and chronic hyperplastic tonsillitis. These patients underwent complete physical examinations including Wassermann and Mantoux tests, thus assuring the absence of any other disease. Postoperatively no reconstituents were prescribed, to avoid altering natural recovery. Fifteen patients of the first group and nine of the second group reported three and four months after the operation for the ptyalin test. The final findings demonstrated that the index of the vital resistance rose considerably after the respective operations. The average increase was 2.60 in the adenoidectomized patients, and 2.22 in the patients whose tonsils and adenoids had been removed. The last physical examination confirmed the results of this test.

SCIARRETTA.

LARYNX

A New Dilator for the Treatment of Acute Corrosive Esophagitis.

Belinoff, Prof. Bronchoscopie, Oesophagoscopie, et Gastroscopie, 1:1 (March), 1940.

In the treatment of acute corrosive esophagitis, the author advocates the use of a specially prepared, sirius coated esophageal bougie, which is resistant to physiological and pathological agents. He feels that the bougie should be inserted immediately following injury to the membranes and be kept in place until complete epithelization has taken place. Its mobility may be maintained by daily partial removal and re-insertion. It is a simple procedure and may be easily performed by the patient.

While this bougie is not considered to be the final answer to the problem of preventing esophageal stenosis, it is felt to be more satisfactory and less hazardous than the daily manipulations of completely inserting and removing other types of bougies.

VIOLE.

The Roentgen Diagnosis of Neoplasms of the Air and Food Passages, With Particular Reference to the Larynx.

Pendergrass, E. P., and Young, B. R. Radiology, 36:197 (Feb.), 1941.

The roentgenographic anatomy of the pharynx, larynx and trachea, including the normal swallowing act, is dealt with in detail, as is the technique for taking roentgenograms in these areas.

Malignant lesions involving one or both pyriform sinuses cause these structures to appear shallow or obliterated.

Extrinsic carcinoma of the larynx may cause thickening of the epiglottis or aryepiglottic folds, best seen in the lateral roentgenogram.

Laryngocele is differentiated from carcinoma involving the ventricle by body-section roentgenograms.

Failure of visualization of a ventricle in the lateral view may be due to a lesion in the ventricle or to extensive ossification of the thyroid cartilage.

Body-section roentgenograms are most important in detecting subglottic extensions of carcinoma of the larynx.

Intrinsic carcinoma or inflammation often causes the arytenoid eminences to stand out more clearly than usual in the lateral view.

This article contains photographs and roentgenograms to illustrate numerous anatomical and pathological structures.

DEAN, JR.

Primary Tuberculosis of the Trachea.

Baron and Lemoine. Rev. de Laryng., Otol., Rhin., (Dec.), 1939.

In a long comprehensive article the authors point out that primary tuberculosis of the trachea is very rare, infection at this site almost always being secondary to pulmonary or laryngeal tuberculosis. When it does occur, it may manifest its presence by an ulceration of the mucosa, granulations, or as a tumor. Tuberculous ulceration of the mucosa is the most frequent lesion and often exhibits no tendency whatever toward cicatrization. From a simple excoriation it develops into an extensive ulcer eroding the tracheal wall down to the cartilage. Sometimes, however, a defense reaction of the mucosa takes place with a spontaneous healing process that surpasses its objective. Granulations overflow the ulceration and protrude into the tracheal tract. The tuberculous tumor rarely remains circumscribed, tending rather to involve the entire tracheal tract and extend into the neighboring tissues. Whatever its evolutionary shape, healing of the lesion may give rise to a stenosing cicatrix.

Dyspnea is always the major functional disturbance dominating the history of the illness. It assumes the particular, pathopneumonic characteristics of tracheal dyspnea. It is apparent at first only on exertion, later becoming continuous and subject to paroxysms. At times it may become so intense as to require emergency treatment for asphyxia.

The prognosis of apparently primary laryngeal tuberculosis is always serious. Quite often the dyspnea becomes progressively worse with fatal results. The prognosis is less grave in the vegetative form which responds to proper treatment. However tracheal localization usually denotes a tuberculous involvement of extreme virulence which may secondarily infect some other organ of the body and cause death.

A diagnosis of primary tracheal tuberculosis is exceptional and may be reached only after eliminating the various causes of tracheal dyspnea by means of minute clinical, radiographic and tracheoscopic

examinations. Final confirmation should be established by the histo-bacteriological examination of a biopsy found to contain the specific tubercular lesions.

Treatment consists of controlling the dyspnea, stimulating cicatrization of the lesion, and improving the patient's general status. Removal of the granulations produces an immediate but temporary feeling of well-being since they have a tendency to recur. Cauterization of the mucosa may result in permanent cicatrization, but the danger lies in the formation of a stenosing cicatrix which would necessitate dilatation later. X-ray treatments are also beneficial and encourage a favorable prognosis. However they should be employed only in the absence of a pulmonary involvement.

Treatment of the patient's general condition should never be neglected and must always remain the basis of any fight against tuberculosis no matter what its form or location.

VIOLE.

Recurring Papillomas of the Larynx.

Galanakis, Emmanuel (Candie, Greece), Rev. de Laryng., Otol., Rhin., 61:13 (April), 1940.

This is a detailed article presenting the author's findings after a study of 315 cases of papillomas of the larynx as reported in both America and Europe. His conclusions are as follows:

1. Papillomas are classified as benign tumors and are of unknown etiology. Still debatable is the theory that they occur as the result of a local or specific inflammation.

2. Clinically two forms are interesting: localized and non-recurring papillomas encountered frequently in adults, and diffuse recurrent papillomas found especially in children. The former are earily cured; the latter return regardless of the method of treatment.

3. The malignant transition of papillomas is very rare. Irritation produced by repeated operations apparently does not give rise to malignant metaplasia.

4. Symptomatic relief and therapeutic measures must be taken.

a. *Symptomatic Relief:* Preferably by tracheotomy, rarely by intubation or emergency removal of the papillomas.

b. *Therapeutic Measures:* Differ from the nonrecurring type in adults and the recurring type in children.

5. The methods proposed include medical, surgical and physical agents.

a. *Medicine* has no appreciable results.

b. *Surgically* the greatest percentage of cures is obtained by laryngostomy, which does not produce stenosis and has no sequelae other than an occasional raucous or hoarse voice.

c. *Physical Agents: Electrocoagulation* by the internal route is dangerous although it may be applied after laryngostomy.

Radium has produced healing in mild cases but its application is considered difficult and dangerous.

X-rays have proven fatal or produced very mediocre results. Recently there has been a tendency toward its use in conjunction with surgery. However, the author considers it a most hazardous method of treatment, especially on children, and advises against its use.

VIOLE.

MISCELLANEOUS

The Use of Histamine in the Treatment of Specific Types of Headaches.

Horton, Bayard T. J. A. M. A., 116:377 (Feb. 1), 1941.

Of 184 cases of headache studied at the Mayo Clinic, 72 fell into a group designated as histaminic cephalgia. The patients in this group had excruciating unilateral pain, involving the eye, temple, neck and often the face, lasting less than an hour and frequently occurring at night. Compression of the common carotid or temporal artery gave temporary relief in some of the cases. Watering and congestion of the eye and nose were noted in many.

The attacks of histaminic cephalgia are shorter than in migraine, lack the hereditary background and have no associated nausea, vomiting or visual disturbances.

In patients who experience this syndrome, attacks can be precipitated by histamine infection. The treatment consists of desensitization, using histamine diphosphate. Maintenance doses are necessary after desensitization has been accomplished.

Histamine desensitization was performed in other types of severe headache and was not found to be effective.

DEAN, JR.